ATLAS OF
SPINE TRAUMA
Adult and Pediatric

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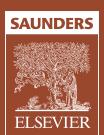## Adult and Pediatric

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Dedication

Daniel H. Kim
To my parents, Chan Taek Kim and Shin Ja Kim

Steve Ludwig
To my wife Cindy, and children Ethan and Katie, who have supported me and allowed me to care for the most emotionally and physically resilient patients, a person with a spinal cord injury.

Alex Vaccaro
I dedicate this book to my only true male hero, my father. I can only dream of being the father, provider and leader he has been for me throughout my life

Jae-Chil Chang
I thank God and wish his blessings on my family (Jeongok, Sunny and Liz), Back-Jang Byun, M.D., Soon-Kwan Choi, M.D., and all others who give their effort to the Neurosurgery Department of the Soonchunhyang University Hospital. This book is dedicated to their neverending support.

Acknowledgments

Preface

Choosing the most appropriate treatment for patients with spinal disorders is a challenging undertaking given the diverse treatment modalities available. Advances in operative techniques and surgical devices have broadened the therapeutic options for spine surgeons. As surgeons, our responsibility is to choose the optimal treatment based on the available literature and focused on optimizing quality of life for our patients.

The treatment of traumatic spinal injuries remains an integral portion of spinal surgery. This text is designed to allow fast and easy reference to surgical procedures and to serve as a practical guide to the spinal surgeon. We hope to give the reader a clear picture of surgical approaches and techniques. Most fundamental approaches from the occipito-cervical junction to the sacroiliac spine are discussed for both adult and pediatric trauma.

It is our hope that this work furthers the understanding of surgical anatomy and operative approaches, and will be an aid to all surgeons in the rapidly changing field of spinal care.

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Epidemiology of Spinal Cord Injury

INTRODUCTION

Spinal cord injury (SCI) represents one the most devastating survivable injuries a patient can suffer. Although SCI often results from a sudden unexpected event, its effects are life changing and result in lifelong constraints and limitations. Effective management of SCI patients requires an understanding of how these injuries occur.

The etiology and incidence of SCI varies by geographic region. As such, many practitioners will never see a patient with a SCI, whereas physicians who work at rehabilitation centers may look at SCI as an epidemic.[1]

Ancient Egyptians felt that SCI could not be meaningfully treated. Hippocrates was the first to treat SCI with the use of traction and immobilization.[2] During World War I, 90% of individuals sustaining a SCI died within 1 year.[3] As recently as the 1940s, SCI was looked on as a death sentence that was fatal, either on its own or through its complications.

Although the life expectancy of a patient with a SCI is still lower than that for the general population, advances in antibiotics, rehabilitation medicine, and medical technology have dramatically improved this number.[4] Currently, more than 95% of patients with acute SCIS survive their initial hospitalization.[5]

The statistics and figures cited in this text are based on the information collected by the National Spinal Cord Injury Statistical Center located at the University of Alabama Birmingham. This database has existed since 1973 and collects data from approximately 13% of new SCI cases in the United States each year through 16 federally funded Model Spinal Cord Injury Care Systems.

INCIDENCE

The estimated annual incidence of SCI in the United States is approximately 40 cases per million population, or 11,000 new cases each year. It has been estimated to affect 1 in 40 patients presenting to a major trauma center. This figure is an underestimate because it does not include patients who die at the scene of an accident. The incidence of fatal SCI before hospitalization, which was previously reported to be as high as 21 cases per million population, is now thought to be as low as 4 cases per million population in some regions.[6–8] Unfortunately, it is not possible to differentiate how much of this decrease is due to improved emergency medical services and how much is due to underreporting.

Although there have been shifts in the underlying causes of SCI, it is not known if the incidence has changed in recent years because there have not been any overall-incidence studies of SCI in the United States since the 1970s.[9] The incidence of SCI outside the United States is consistently lower than within the country. Many experts attribute this difference to the increased incidence of violence-related SCI in the United States. Violence is an uncommon cause of SCI in other countries.[10–13]

PREVALENCE

Several authors have attempted to calculate the prevalence of SCI in the United States.[14–16] In the early 1980s DeVivo et al.[14] estimated the prevalence of SCI using the available data at that time on incidence and life expectancy. They reported the prevalence of SCI to be 906 patients per million population, or approximately 200,000 patients. This was probably an overestimate based on the fact that increasing life expectancy was not accounted for.

Berkowitz et al.[15] used a probability sampling plan of small geographic areas in the late 1980s and estimated the prevalence of SCI to be 721 patients per million population, or 177,000 cases. Lasfargues et al.,[16] using a mathematical model, combined Berkowitz's findings with estimates of age- and gender-specific incidence and mortality and estimated that there would be more than 276,000 patients with SCI in the United States by 2014. This increase in prevalence is almost exclusively the result of improved life expectancy. According to the National Spinal Cord Injury Statistical Center (NSCISC) there are approximately 250,000 spinal cord injured patients living in the United States as of July 2005.

AGE AT INJURY

SCI occurs most commonly in people between 16 and 30 years of age. These injuries occur more commonly in this age group than all other age groups combined. The mean age of patients with SCI is 33 years, a number that has increased steadily over the last 20 years. Table 1-1 reflects a consistent trend toward older age at time of injury. Mean age at injury has increased from 28.7 years between 1973 and 1979 to 37.6 years since 2000. During that same time the average age of the U.S. population also has increased.

According to the NSCISC the most common age of patients with SCI is 19 years. Thirty percent of spinal cord–injured patients are between the ages of 17 and 23 years, 52% are between the ages of 16 and 30 years, and 9% are older than 60 years.

The ages of SCI patients at the time of injury (incident cases) is different from the current ages of patients living with a SCI (prevalent cases) because the number of prevalent cases is a function not only of age at injury but also of long-term survival. In 1988 Berkowitz et al.[15] attempted to estimate the ages of all patients living in the United States with a SCI. At that time the median age of all patients living with a SCI in the United States was 41 years old, with more than half of the patients between the ages of 25 and 44 years.

GENDER

The percentage of male patients in the Spinal Cord Injury Database is 81.1%. The percentage of SCI patients living in the United States today that are men is much lower than the percentage of new cases that are men because of the difference in survival rates between men and women. In the Berkowitz et al. study,[15] 71% of prevalent cases were men, a figure that would be even lower today because the life expectancy of women is greater than that of men.

ETHNICITY

When evaluating SCI patients by race, it is important to realize that there is a great amount of variability between model systems. The proportion of Caucasian patients ranges from 22.6% to 89.7%, whereas the proportion of African-American patients ranges from 3.6% to 52.1%. The highest reported proportion of Native American patients and Asian patients is 2.7% and 5.6%, respectively.

Between 1973 and 1979, 76.8% of patients enrolled in the database were Caucasian, 14.2% were African American, 1.9% were Native American, and 0.9% were Asian. However, since 1994 the number of Caucasian patients enrolled in the database has decreased to 66.1%, while the number of African-American patients has increased to 25.3%, and 2.9% of enrolled patients are classified as other races. This trend is due in very small part to changes in the general U.S. population. It is due, more so, to periodic changes in the identities and locations of model systems, changes in eligibility criteria for inclusion in the database, and changes in referral patterns to model systems.

Because several studies have shown conclusively that the incidence of SCI is most common among non-Caucasians,[17] it should not be concluded from the database that the incidence of SCI is more common among Caucasians than non-Caucasians. It should be understood that most patients in the database are Caucasians because this race group encompasses the largest segment of the U.S. population.

ETIOLOGY

The individual causes of SCI are grouped into five categories for analytical purposes. These categories are vehicular accidents, violence, recreational sports, falls, and other causes. Vehicular accidents are the leading cause of SCI in the United States (43.4%), followed by falls (19.7%), and acts of violence (18.2%).

The three leading causes of SCI among men and women are motor vehicle accidents, falls, and gunshot wounds, respectively. Diving accidents and motorcycle crashes rank fourth and fifth for men, respectively. The fourth and fifth leading causes of SCI among women are medical or surgical complications and diving accidents, respectively. Automobile accidents and medical and surgical complications account for a significantly lower percentage of SCIs among men than women. On the other hand, a significantly higher percentage of men, as compared with women, contract SCIs from diving accidents, from motorcycle accidents, and from being hit by falling objects.

Figure 1-1 depicts grouped etiology by gender. As stated earlier, 81.1% of all SCIs were incurred by men. The sports category differs the most from this overall distribution. In this category 89.8% of injuries were incurred by males.

TABLE **1-1**   **Mean Age of Spinal Cord Injury (SCI)**

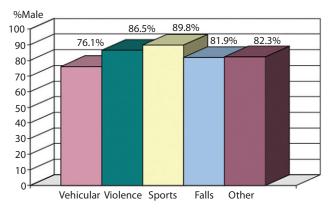| Year of Injury	Mean	Standard Deviation	n	Minimum	Maximum
1973–1979	28.7	14.1	4564	1	88
1980–1984	30.5	14.6	4951	1	90
1985–1989	32.3	15.8	3842	<1	92
1990–1994	33.7	15.9	3296	1	97
1995–1999	36.4	16.9	3624	<1	98
2000–2005	37.6	16.7	3405	4	90

Fig. 1-1 Grouped etiology by gender.

According to the NSCISC database, diving accidents account for the most SCIs secondary to recreational sports (6.8% overall).[18] Snow skiing, football, ATV, and horseback riding all rank a distant second (0.5% overall). The incidence of SCI in football has decreased because of education regarding proper tackling techniques and the use of better equipment.

Grouped causes of SCI by age, depicted in Figure 1-2, show that among patients younger than the age of 60 years, vehicular accidents are the leading cause of SCI. Falls are the leading cause of SCI after the age of 60 years. With advancing age, sports accidents and violence account for a smaller proportion and falls account for a larger proportion of SCI.

Other than SCIs secondary to acts of violence, the most common causes of SCI in other countries are similar to those seen in the United States.[11] Specifically with regard to acts of violence, suicide accounts for approximately 80% of SCIs resulting from acts of violence in other countries. In the United States, violence that results in SCI is more commonly malicious in nature. Analysis of the interaction between age, gender, race, and etiology shows the rather disturbing trend that nearly 70% of SCIS in African-American and Hispanic males between the ages of 16 and 21 is due to acts of violence. The comparable figure for Caucasian males in this same age group is less than 8%.

TIME OF INJURY

Traumatic SCI, similar to other injuries, occurs more often on weekends than weekdays. According to the NSCISC database, nearly 20% of SCIs occurred on Saturday, and approximately 17% occurred on Sunday. Tuesdays and Wednesdays are the least likely days for a SCI to occur at 11%, respectively.

The incidence of SCI also varies by season, peaking in July (10%) and being at its lowest in February (6.5%). This seasonal variation is more pronounced in the northern United States where there is greater seasonal variation in the climate. The greater incidence of SCI in the summer months is directly related to increased motor

vehicle–related and diving and other recreational sports injuries.

ASSOCIATED INJURIES

SCIs are accompanied by other significant injuries just under 50% of the time.[19] The most often associated injuries are to the trunk, which can be related either to gunshot injuries or to high-energy trauma.[20]

There is an association between the nature and frequency of associated injuries and the etiology of the SCI. SCI caused by motor vehicle accidents is associated most often with loss of consciousness (43%), followed by fractures of the trunk or long bones (40%), and then head injury (18%) and traumatic pneumothorax (17%). On the other hand, patients who contract their SCI during a sports accident will have associated loss of consciousness 22% of the time. Those who are injured by acts of violence most commonly have an associated traumatic pneumothorax (36%) and rarely have any other associated injuries.

The management of these and other associated life-threatening injuries often supersedes the management of the SCI. Emergency surgery for trauma to other areas can delay diagnosis and surgical management of the spine injury.[21] It is vital to manage shock and hypoperfusion appropriately in the face of associated injuries to optimize spinal cord recovery.
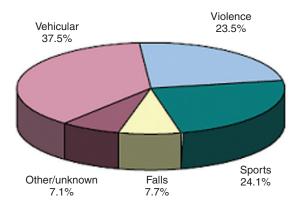
NEUROLOGIC LEVEL AND EXTENT OF INJURY

The neurologic level of SCI is defined as the lowest level of the spinal cord that has intact motor and sensory function bilaterally. According to the NSCISC database, at discharge 51% of SCI patients have cervical lesions, 34% have thoracic lesions, and 10% have lumbosacral lesions. The most common level of lesion on discharge is C5 (15%), followed by C4 (14%), C6 (11%), and T12 (7%).
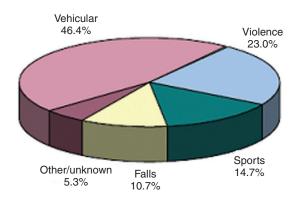
Neurologic impairment refers not only to the neurologic level but also to the extent of injury. At discharge, neurologically incomplete tetraplegia is most common (30%), followed by neurologically complete paraplegia (26%), neurologically complete tetraplegia (21%), and neurologically incomplete paraplegia (18.5%).

There is a strong correlation between etiology of SCI and neurologic category. Neurologically incomplete tetraplegia ranked first for all etiologies (as discussed previously) except for acts of violence in which neurologically complete paraplegia is most common.
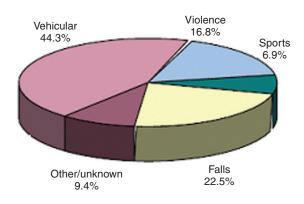
The proportion of patients with neurologically incomplete lesions at discharge has increased from 46% in the 1970s to 55% currently. This is due in part to improved emergency medical services. Despite the advent of high-dose methylprednisolone therapy, the number of neurologically incomplete lesions at the time of discharge

Vehicular
37.5%

Violence
23.5%

Sports
24.1%

Falls
7.7%

Other/unknown
7.1%

0 to 15 Years

Vehicular
46.4%

Violence
23.0%

Sports
14.7%

Falls
10.7%

Other/unknown
5.3%

16 to 30 Years

Vehicular
44.3%

Violence
16.8%

Sports
6.9%

Falls
22.5%

Other/unknown
9.4%

31 to 45 Years

Vehicular
38.3%

Violence
8.4%

Sports
3.3%

Falls
36.8%

Other/unknown
13.3%

46 to 60 Years

Vehicular
31.8%

Violence
3.2%

Sports
2.1%

Falls
48.0%

Other/unknown
15.0%

61 to 75 Years

Other/unknown
9.7%

Vehicular
27.6%

Violence 1.6%

Sports 0.5%

Falls
60.7%
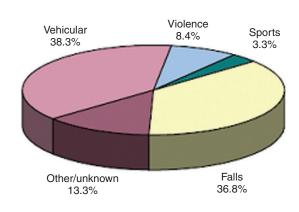
76 to 98 Years

Fig. 1-2 Grouped etiology by age at injury.

decreased between 1991 and 1994 at the same time that SCI secondary to acts of violence increased. This is most likely because SCI caused by gunshot wounds is almost always complete. Since 1994, the percentage of incomplete injuries has again increased as the number of SCIs secondary to acts of violence has decreased.

MARITAL STATUS

Given the fact that most SCIs occur in young people, it follows that more than 50% of patients enrolled in the NSCISC database have never been married at the time of their injury. Unfortunately, the annual marriage rate after SCI is almost 60% below that of the general population.[22] The annual divorce rate during the first 3 years after SCI is 2.5 times normal. The divorce rate is nearly twice as high for those marriages that occur after SCI.[23,24]

LEVEL OF EDUCATION

Although nearly 90% of SCI patients in the NSCISC database are 19 years of age or older and would be expected to have completed high school, only 58% of enrolled patients are high school graduates at the time of admission. Nearly 10% of SCI patients have an eighth-grade education or less.

Among patients enrolled in the database who were in the 9th to 11th grade at the time of their injury, only 46.5% obtained a high school diploma within 5 years after injury. Of enrolled patients who had completed high school at the time of injury, 12.9% completed a post–high school degree within 5 years.
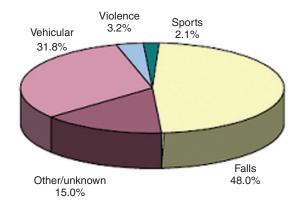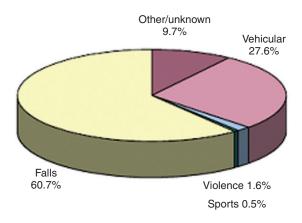
OCCUPATIONAL STATUS

At the time of injury, nearly two thirds (64%) of SCI patients are employed in the competitive labor market while 15% are students and 16.4% are unemployed. The unemployment percentage is greater than the typical unemployment rate of the general population of the United States. At 1-year postinjury only 13.6% of SCI patients are employed. This number peaks at 3-years postinjury to just over 44%.

Factors associated with an increased likelihood of postinjury employment include younger age, Caucasian race, male gender, higher level of education, less severe injury, being employed at the time of injury, having a nonviolent injury etiology, being able to drive, and greater elapsed time after injury.[25,26] Patients who are able to return to work within 1 year of injury usually will return to the same job withthe same employer. Patients who return to work more than 1 year after their injury usually obtain a different job with a different employer. According to Young et al.[27] patients with SCI most commonly acquire either professional/technical jobs or clerical/sales jobs.

DISCHARGE PLACEMENT

Most patients enrolled in the NSCISC database (88.1%) are discharged to a private residence. Less than 1% of patients are discharged to another acute care hospital and only 4% are discharged to a nursing home.

Factors that predict discharge to a nursing home include cervical injury without useful motor function capability below the injury level, ventilator-dependence, older age, being unmarried, being unemployed, and having either Medicaid or health maintenance organization (HMO) insurance.[28]

According to the NSCISC database, nearly 98% of patients who are still alive 10 years after injury reside in private residences in the community. Not surprisingly, age is the most significant predictor of long-term nursing home residence with more than 20% of patients greater than 75 years of age residing in a nursing home at the time of their most recent injury anniversary.[29]

LIFE EXPECTANCY

Although the life expectancy of SCI patients has improved over the past few decades, it remains below that of the noninjured population. Patients who are admitted to a model system within 24 hours of injury have an estimated mortality rate of approximately 6% in the first year. This number declines to nearly 1% per year after the second postinjury year.

Factors associated with increased risk of mortality during the first year after injury include advanced age, male gender, injury level at C4 or above, ventilator-dependence, having a neurologically complete injury, and being injured by an act of violence. It should be noted that there has been a significant decline in first-year mortality rates over time. The risk of patients dying within the first year after injury is nearly 70% less today than it was for patients injured in the 1970s.[30]

Aside from the factors listed previously, predictors of mortality in later years after SCI include poor health, emotional distress, and poor self-rated adjustment to disability. The substantial declines seen in mortality rates during the first year after injury have not been observed in following years.

Tables 1-2 and 1-3 outline the latest life expectancy estimates from the NSCISC database. It should be noted that life expectancy is almost normal for patients with incomplete motor injuries but declines steadily with increasing injury severity. Similar tables have been produced for patients with SCIs in other countries showing comparable life expectancies in those countries as compared with the United States.

COMPLICATIONS

Pneumonia is the most common medical complication affecting SCI patients, occurring in 34% of patients. Pressure ulcers (33.6%), deep vein thromboses (15%), and pulmonary emboli

TABLE 1-2   Life Expectancy for Spinal Cord Injury (SCI) Persons Surviving at Least 24 Hours Postinjury

		LIFE EXPECTANCY (YEARS)				
		NOT	VENTILATOR	DEPENDENT		VENTILATOR
AGE AT INJURY (YEARS)	NO SCI	MOTOR FUNCTION ANY LEVEL	PARAPLEGIA	TETRAPLEGIA C5-C8	TETRAPLEGIA C1-C4	DEPENDENT ANY LEVEL
10	67.9	62.2	54.7	49.4	44.8	23.2
15	63	57.3	49.8	44.6	40.0	19.0
20	58.2	52.6	45.3	40.2	35.9	16.4
25	53.5	48.0	40.9	36.1	31.9	14.3
30	48.7	43.3	36.4	31.8	27.9	11.8
35	44.0	38.7	32.0	27.6	23.8	9.2
40	39.3	34.1	27.7	23.5	20.0	6.9
45	34.8	29.8	23.6	19.7	16.5	5.1
50	30.3	25.5	19.8	16.2	13.3	3.6
55	26.1	21.5	16.1	12.9	10.3	2.4
60	22.0	17.7	12.8	10.0	7.8	1.4
65	18.2	14.2	9.9	7.4	5.6	0.6
70	14.7	11.1	7.4	5.3	3.8	0.1
75	11.5	8.3	5.2	3.6	2.5	<0.1
80	8.8	6.0	3.5	2.2	1.4	<0.1

TABLE 1-3   Life Expectancy for Spinal Cord Injury (SCI) Persons Surviving at Least 1-Year Postinjury

		LIFE EXPECTANCY (YEARS)				
		NOT	VENTILATOR	DEPENDENT		VENTILATOR
AGE AT INJURY (YEARS)	NO SCI	MOTOR FUNCTION ANY LEVEL	PARAPLEGIA	TETRAPLEGIA C5-C8	TETRAPLEGIA C1-C4	DEPENDENT ANY LEVEL
10	67.9	62.9	55.3	50.6	46.8	30.9
15	63.0	58.0	50.5	45.8	42.1	26.4
20	58.2	53.2	45.9	41.4	37.8	23.1
25	53.5	48.6	41.5	37.2	33.8	20.1
30	48.7	43.9	37.0	32.9	29.6	17.1
35	44.0	39.3	32.6	28.6	25.5	13.9
40	39.3	34.7	28.3	24.4	21.5	10.9
45	34.8	30.3	24.1	20.6	17.9	8.4
50	30.3	26.0	20.3	17.0	14.5	6.3
55	26.1	22.0	16.6	13.6	11.4	4.5
60	22.0	18.1	13.2	10.6	8.7	3.0
65	18.2	14.6	10.3	8.0	6.4	1.8
70	14.7	11.4	7.7	5.8	4.5	1.0
75	11.5	8.6	5.5	3.9	2.9	0.3
80	8.8	6.2	3.7	2.5	1.7	<0.1

(3.6%) follow, respectively. Less than 20% of SCI patients had no secondary medical complications.

Patients with neurologically complete tetraplegia are at the highest risk for all secondary medical complications except deep vein thromboses and postoperative wound infections. According to the database, 60% of these patients developed pneumonia, 53.2% developed pressure ulcers, 16.1% developed deep vein thromboses, and 5.3% developed a pulmonary embolus.

OPERATIVE PROCEDURES

During system hospitalization more than half the patients underwent a spinal fusion and almost half had internal fixation procedures performed. Operations that were performed on more than 10% of the patients included placement of traction and/or halo, spinal decompression, and laparotomy.

During postinjury years, closure of pressure ulcers was the most commonly performed procedure in all years except

year 25 when calculus removal ranked first. For all years except year 25, calculus removal was the second-most common procedure performed.

CAUSE OF DEATH

Primary causes of death for all patients enrolled in the NSCISC database are listed in Table 1-4. Diseases of the respiratory system are the leading cause of death among patients with SCI. Nearly 72% of these are secondary to pneumonia. Unexplained myocardial infarction and infective and parasitic diseases rank second and third, respectively. With regard to infection, 93% of cases were secondary to septicemia associated with decubitus ulcers, urinary tract infections, or respiratory tract infections. Hypertensive and ischemic heart disease was the fourth-leading cause of death. Neoplasms ranked fifth: the most common locations being lung (28.8%), bladder and colon/rectum (6.9%), and prostate (5.8%).

It is important to compare the causes of death in the SCI population to those of the general population. It has been estimated that deaths because of septicemia, diseases of the pulmonary system, and pneumonia occur far more often in patients with SCIs. On the other hand, mortality secondary to ischemic heart disease and cancer is equally frequent in patients with and without SCI.

COST OF SCI

The pressures of cost containment have affected the care of SCI patients. The mean total system charges for patients with SCI increased from $156,000 in 1973 to $282,000 today. According to the NSCISC database, mean acute care length of stay has declined from 28 days in 1973 to 19 days in 2003. Mean rehabilitation length of stay has declined from 115 days in 1974 to only 45 days in 2003. This is in contrast with Europe, where acute care length of stay continues to average approximately 2 months and the average rehabilitation stay is 7 months.[11]

A recent study attempted to estimate the first year and annual expenses for patients with SCI over their remaining lifetime by neurologic level and extent of injury.[31] Average first-year expenses ranged from more than $700,000 for patients with upper cervical spine injuries (C1-C4) to $210,000 for patients with incomplete motor injuries at any level. Average annual charges for the remainder of life for these two groups were estimated to be $130,000 and $15,000, respectively.

Categorization of these charges reveals that during the first year most charges are due to inpatient acute care and rehabilitation costs. Recurrent annual charges, on the other hand, are for attendant care, rehospitalizations, durable equipment, outpatient services, and medications.[31]

TABLE 1-4   Primary Cause of Death in Patients With Spinal Cord Injury

ICD9CM CODES	PRIMARY CAUSE OF DEATH	n	%
460-519	Diseases of the respiratory system	782	21.9
420-429	Other heart disease	445	12.4
000-139	Infective and parasitic diseases	338	9.4
400-414	Hypertensive and ischemic heart disease	281	7.9
140-239	Neoplasms	260	7.3
E800-E949	Unintentional injuries	194	5.4
415-417	Diseases of pulmonary circulation	179	5.0
520-579	Diseases of the digestive system	171	4.8
780-799	Symptoms and ill-defined conditions	161	4.5
E950-E959	Suicides	135	3.8
430-438	Cerebrovascular disease	133	3.7
580-629	Diseases of the genitourinary system	131	3.7
E980-E989	Subsequent trauma of uncertain nature (unintentional/suicide/homicide)	118	3.3
320-389	Diseases of the nervous system and sense organs	59	1.6
240-279	Endocrine, nutritional, metabolic, and immunity disorders (includes AIDS)	54	1.5
440-448	Disease of the arteries, arterioles, and capillaries	53	1.5
E960-E969	Homicides	35	1.0
290-319	Mental disorders	11	0.3
451-459	Diseases of veins, lymphatics, and other diseases of the circulatory system	10	0.3
710-739	Diseases of the musculoskeletal system and connective tissue	9	0.3
740-759	Congenital anomalies	5	0.1
Residual	All others	4	0.1
280-289	Diseases of blood and blood-forming organs	4	0.1
E970-E979	Legal intervention	2	0.1
	Total known causes of death	3574	
	Total unknown causes of death	3491	
	Total deaths	7065	

Average first year and annual charges for SCI have also been grouped based on etiology. Not surprisingly, first year and annual recurring charges are highest for SCIs that are the result of sports mishaps because they usually result in cervical SCI and motor vehicle accidents because they often involve other injuries in addition to the SCI.

CONCLUSION

By studying the epidemiology of SCI, the physician not only can make predictive assumptions but also can direct his or her efforts toward the prevention of SCI and its secondary problems.

Through the information gathered by the NSCISC database, as well as state registries and studies from inside and outside the United States, we can continue to make advances such as the significant improvement in survival of SCI patients.

Future efforts should be directed toward improved data collection and improved clinical services to allow for near-normal postinjury lives.

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C H A P T E R

2

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Anatomy and Pathophysiology of Spinal Cord Injury

INTRODUCTION

The annual incidence of spinal cord injury in developed countries ranges from 11.5 to 53.4 per million population.[1,2] There are many etiologies of spinal cord injury with motor vehicle accidents, the most common cause of spinal cord injury in the United States. Other causes include recreational activities, violence, and work-related injuries.[3] Spinal cord injury predominantly affects young males and there is significant cost to the patient, the patient's family, and society. Moreover, there is considerable psychological cost to the patient and family.

Despite approximately four decades of research focusing on the pathophysiology of spinal cord injury, there is still a lack of a full understanding of the cellular, biochemical, and molecular changes that occur following injury and how to overcome them with pharmacologic therapy. Although the use of methylprednisolone (intravenous [IV]) demonstrated a mild benefit when delivered within 8 hours of spinal cord injury,[4] methodologic concerns regarding the human studies have led many to suspect the true benefit of this medication.[5] Despite such skepticism, IV methylprednisolone remains the only pharmacologic therapy with any claim of efficacy for patients with spinal cord injury.

The purpose of this chapter is to review the anatomy of the spinal cord and the pathophysiology of spinal cord injury. This information is necessary for proper management and to develop new treatment strategies for patients that have sustained a spinal cord injury.

ANATOMY

A detailed description of spinal cord anatomy is beyond the scope of this chapter. We focus on the clinically relevant anatomy of the spinal cord as it relates to spinal cord injury and present a number of spinal cord injury syndromes.

The spinal cord begins as the termination of the medulla oblongata and continues to end as the conus medullaris at approximately L1-L2 in adults. Nerve roots continue on as the cauda equina. The spinal cord is covered by pia, arachnoid, and dura mater. The latter continues down to the sacrum. The spinal cord's blood supply is derived from one anterior and two posterior spinal arteries and a number of radicular branches along the length of the spinal cord. These radicular feeders reach the spinal cord by passing along the spinal nerve and then the posterior and anterior rootlets, more so with the posterior than the anterior arteries.

In cross section, the spinal cord consists of central gray matter and peripheral white matter (Fig. 2-1). The gray matter mainly contains neurons, with the anterior horn containing motor neurons, and the posterior gray matter with interneurons concerned with the reception and transmission of sensory information. The intermediate columns are concerned with the autonomic nervous system. The gray matter receives a considerably greater amount of blood supply compared with the white matter.

The gray matter is a butterfly-shaped structure in the center of the spinal cord. It is surrounded completely by white matter. Rexed organized the spinal gray into 10 lamina based on cytoarchitecture. Although Rexed studied these laminae in the cat spinal cord, it is assumed that the same pattern exists in humans. These laminae constitute regions with characteristic cytologic features in which there are differences at various segmental levels of the spinal cord. The boundaries between the laminae may occur gradually or abruptly.[6]

The white matter of the spinal cord is organized into three funiculi: the dorsal funiculus, the lateral funiculus, and the ventral funiculus. Each funiculus is composed of one or more tracts.

DORSAL FUNICULUS

The dorsal funiculus contains the ascending fasciculi gracilis and cuneatus. These tracts convey proprioceptive, vibratory, and discriminative touch information. The fasciculus gracilis
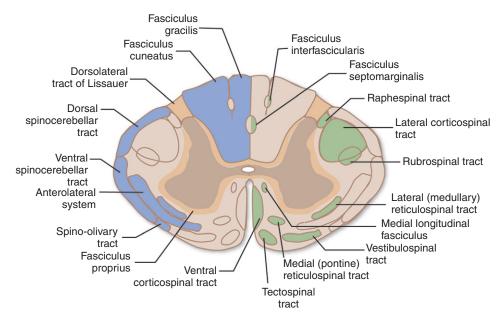
Fig. 2-1 Cross section of the spinal cord. (Reprinted with permission from Cramer GD, Darby SA: Basic and clinical anatomy of the spine, spinal cord, and ANS, St Louis, 1995, Mosby).

is more medially located in the spinal cord and contains primarily cervical afferent axons whereas the fasiculus cuneatus is located lateral and has lumbar and sacral tracts. Fibers entering below T6 and thus subserving the lower extremity are situated medially in the cord (i.e., in the fasciculus gracilis), whereas those entering above T6, upper extremity, are situated more laterally (cuneatus).

The primary neuron cell body is located in the dorsal root ganglion (DRG). Its axon enters the spinal cord and ascends, terminating in its respective nuclei (gracilis or cuneatus). Secondary axons then cross as internal arcuate fibers and ascend the brainstem through the medial lemniscus. These axons terminate in the posterolateral thalamus. Tertiary neurons then enter the primary sensory cortex. This is by far an oversimplification, with the dorsal columns being much more complex. Lesions of the dorsal funiculus lead to a loss or diminution of vibratory sense, position sense, two-point discrimination, touch, and weight perception. Sensory thresholds and fine manipulative movements of the digits also may be affected. The effect is ipsilateral if the lesion is in the spinal cord and contralateral with a brainstem or thalamic lesion.

VENTRAL AND LATERAL FUNICULI

As opposed to the dorsal funiculus, the ventral and lateral funiculi contain several ascending and descending tracts.

LATERAL SPINOTHALAMIC TRACT

This tract is situated in the lateral funiculus. It is concerned with the transmission of pain and thermal sensations and transmits light touch. Both unmyelinated and thinly myelinated fibers contribute to the tract and have their cell bodies in the DRG. Because these fibers are unmyelinated and thinly myelinated respectively, they occupy a lateral position in the dorsal root. Axons enter the spinal cord and ascend one or two levels prior to synapsing in the dorsal horn. Secondary axons cross to the contralateral side through the anterior white commissure and ascend the spinal cord in the lateral spinothalamic tract. The fibers then synapse in the ventral posterolateral thalamus and continue on to the primary sensory cortex.

The tract is somatotopically arranged so that those axons relaying information about cervical levels are most medial, while those from sacral levels are most lateral. The unique somatotopic organization may aid in the localization of certain spinal cord lesions. For example, a central lesion may present initially with upper extremity weakness. The paresis may then descend as the lesion enlarges. In contrast, an extrinsic compressive lesion may present initially with lower extremity weakness and the paresis will often ascend. Midline, ventral lesions may interrupt the ventral white commissure, leading to bilateral loss of pain and temperature sensation, often in a capelike distribution. This is often seen in syringomyelia. Lesions in the spinal cord, brainstem, or brain will result in contralateral loss of pain and temperature sensation below the lesion.

CORTICOSPINAL TRACT

This tract is necessary for fine motor movements, especially fine finger movements. Axons originating in the primary motor, premotor, and sensory cortex make up the corticospinal tract. Fibers descend from the cortex forming the corona radiate, internal capsule, cerebral peduncle, and finally enter

the medulla. The fibers are situated on the ventral medulla and form the large pyramids. Here they cross at the pyramidal decussation to descend in the cord as the lateral corticospinal tract (majority of fibers). This tract exists in the lateral funiculus. Some uncrossed fibers form the ventral corticospinal tract, which is situated in the ventral funiculus. The lateral corticospinal tract is somatotopically arranged, with cervical fibers situated medially and sacral fibers most laterally. One may understand how a slowly expanding intramedullary mass in the cervical spinal cord may first lead to upper extremity weakness followed by a descending paralysis.

Axons from the corticospinal tract terminate directly on motor neurons or interneurons in the ventral gray matter of the spinal cord. These then exit the cord as motor neurons in the ventral root. Lesions of the corticospinal tract initially lead to paralysis, which eventually evolves into a set of "upper motor neuron" signs. These signs include spasticity, exaggerated deep tendon reflexes, Babinski sign, and clonus.

There are many other tracts, both ascending and descending, in the spinal cord that contribute to normal neurologic functioning. These include the spinocerebellar tracts, the reticulospinal tracts, rubrospinal, vestibulospinal, and other tracts. Lesions of these tracts may be responsible for some of the more subtle signs of spinal cord injury and are associated with particular syndromes when damaged. Manifestations of damage to these tracts are usually detected in patients with demyelinating, degenerative, or congenital disorders. A full discussion of such syndromes is beyond the scope of this chapter, which focuses on spinal cord injury. An excellent overview of such tracts may be found in *Core Text of Neuroanatomy.*[7]

BLOOD SUPPLY

The blood supply to the spinal cord is provided by one anterior spinal artery and paired posterior spinal arteries (Fig. 2-2). The posterior cerebral circulation, paired vertebral arteries, and/or posterior inferior cerebellar arteries are the origin of the anterior and posterior spinal arteries. A radicular artery accompanies the nerve root at every spinal level. These actually contribute very little to the spinal cord itself.
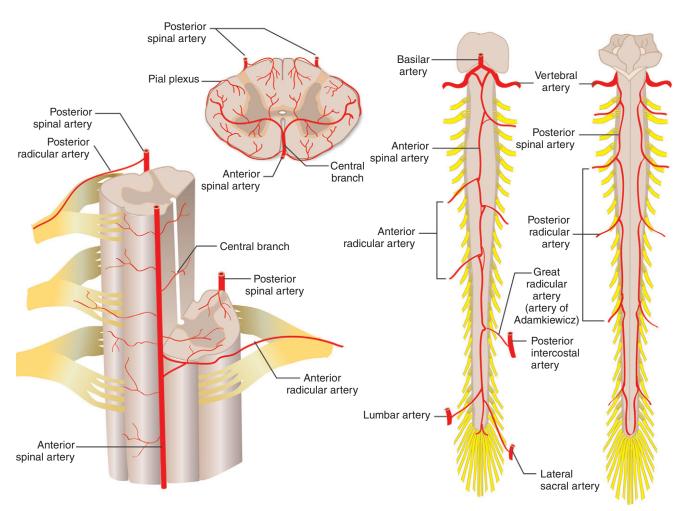

Fig. 2-2 Arterial supply of the spinal cord. (Reprinted with permission from Cramer GD, Darby SA: Basic and clinical anatomy of the spine, spinal cord, and ANS, St Louis, 1995, Mosby).

The posterior spinal arteries are less well-defined than the anterior spinal artery and are fed by 10 to 23 radicular branches. The anterior spinal artery is fed by only six to eight radicular arteries and the blood supply from the anterior spinal artery is therefore somewhat more tenuous than that from the posterior spinal arteries. These radicular arteries tend to be somewhat constant and include a branch from the vertebral artery at around C3, a branch from the deep cervical artery around C6, a branch from the costocervical trunk at about C8, a high thoracic radicular artery and the artery of Adamkiewicz. The artery of Adamkiewicz is the main supply to the anterior spinal cord below approximately T8; it is usually located on the left. These limited contributions to the anterior system leave a "watershed zone" in the mid-thoracic region, which is susceptible to infarction.[8]
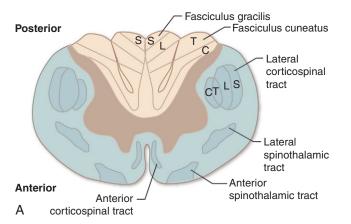
SPINAL CORD SYNDROMES

Injury to the spinal cord results in either a complete or incomplete spinal cord injury. Patients with incomplete injuries have some function below the level of injury and a number of syndromes have been described that are related to the specific spinal cord anatomy described earlier in this chapter.
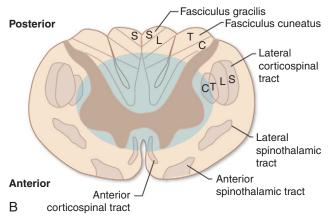
ANTERIOR CORD SYNDROME (Fig. 2-3, *A*)

This syndrome is often due to the occlusion of the anterior spinal artery. There is infarction of the spinal cord in the territory supplied by the anterior spinal artery, which is the anterior and lateral funiculi. Therefore, the posterior funiculus is preserved. Patients display flaccid paralysis below the lesion and loss of pain and thermal sensation because of disruption of the corticospinal and spinothalamic tracks bilaterally. There is preservation of proprioception, two-point discrimination, and crude sensation (i.e., pressure) because of preserved dorsal column function. Prognosis for recovery of anterior cord function is poor.

CENTRAL CORD SYNDROME (Fig. 2-3, *B*)

This syndrome typically occurs in the older population with preexisting spinal stenosis. There is often minor trauma and the patient presents with weakness and numbness primarily affecting the upper extremities and less so the lower extremities. This has been described as a "man in a barrel" phenomenon. The pathologic lesion, either edema and/or hemorrhage, occurs in the central gray matter and can extend to the more centrally located axonal tracts of the spinal cord. As can be seen from the topography of the major motor and sensory tracks, the cervical regions are most medial with the lumbar and sacral regions most lateral. This explains why the weakness and numbness primarily affects the upper and not the lower extremities, at least early on in the disease process.[9] The central region of the spinal cord, in the author's opinion, may be viewed as a watershed zone. Vessels must travel a long distance between the surface and the most ventral gray matter, which may explain why the central region of the cord is affected
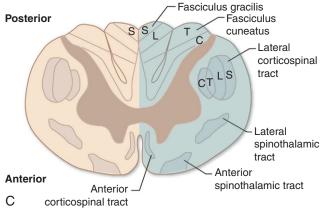
Fig. 2-3 Incomplete spinal cord syndromes. *A,* Anterior cord syndrome, *B,* Central cord syndrome, *C,* Brown-Séquard syndrome. (Reprinted with permission from Browner BD, Jupiter JB, Levine AM, et al. [eds]: Skeletal Trauma: Basic Science, Management and Reconstruction, 3rd ed, vol 1. Philadelphia, Saunders, 2003.)

preferentially over lateral white matter in this type of injury. The prognosis for recovery is generally good.

BROWN-SÉQUARD SYNDROME (Fig. 2-3, *C*)

In this condition, there is a hemi-section of the spinal cord, either through direct transection or functionally via compression from a laterally situated mass. This affects the dorsal

columns, corticospinal, and spinothalamic tracts on the ipsi-lateral side of the spinal cord. The resultant clinical picture is that of ipsilateral weakness, ipsilateral loss of proprioception and vibration, and contralateral loss of pain and thermal sensation below the lesion. There is loss of pain and thermal sensation on the ipsilateral side only at the level correspond-ing to the site of the lesion. Patients with Brown-Séquard syndrome usually have the best chance of recovery of all in-complete lesions.

PATHOPHYSIOLOGY OF SPINAL CORD INJURY

It is generally accepted that spinal cord injury involves pri-mary and secondary mechanisms of injury.[10,11] The primary injury is the inciting event leading to the spinal cord injury. The primary injury is generally because of deformation of the spinal cord and possibly continued compression.[12] Ex-amples of primary injury include acute herniated disk, burst fracture, fracture dislocation, and so forth. As can be seen, there is an initial deformation of the cord because of the injury and a continued compression because of the disk, fracture fragment, and/or deformity. Continued com-pression may not occur when there is dislocation and re-duction prior to presentation to the hospital and imaging performed.

The concept of secondary injury following spinal cord injury was first introduced by Allen.[13] While studying a hem-orrhage in the spinal cord of dogs, he noted that a "factor" present in the hemorrhage may be responsible for ongoing damage, so-called secondary injury.

Prevention and perhaps protective equipment is essen-tially the only way to limit the primary injury in spinal cord injury. Excellent efforts by public education, key leg-islation, modifications in sporting rules and newer re-straints systems in automobiles have decreased incidences of spinal cord injuries from certain causes. However, once an injury has occurred nothing can be done presently to change the irreversible neural injury from the primary in-sult. Attention must be directed at limiting the secondary injury.

SECONDARY INJURY

The primary injury initiates a cascade of events that is known as secondary injury (Fig. 2-4). Many types of secondary in-jury mechanisms exist, at least in theory or in the experimen-tal animal. The number of mechanisms can be divided into cellular, biochemical, vascular, and molecular events. The fi-nal common pathway is ultimately cellular death and axonal necrosis (Fig. 2-5).

VASCULAR CHANGES

Vascular changes following spinal cord injury can be divided into local and systemic. Immediately following spinal cord injury there is an initial increase in systemic blood pressure
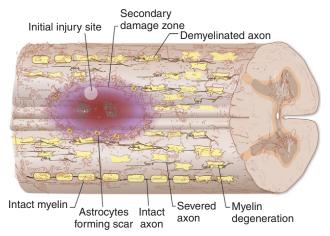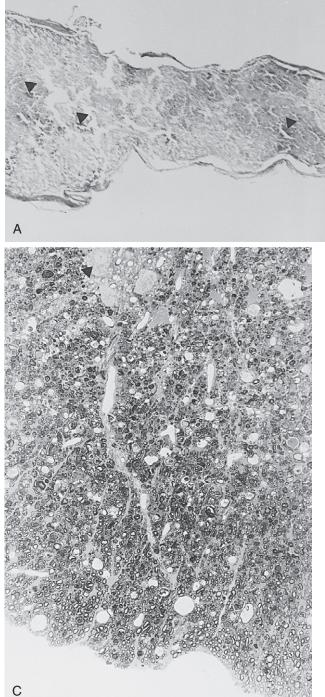

Fig. 2-4 Illustration of events occurring following spinal cord injury. Early after injury, the spinal cord may appear grossly normal although swelling may be apparent. Mi-croscopically, there may be areas of edema, hemorrhage, and myelin destruction. (Reprinted with permission from the Spinal Cord Tutorial on www.CristopherReeve.org.)

followed by hypotension.[14–19] The hypotension is related to neurogenic shock.[20] There is decreased systemic vascular re-sistance related to loss of sympathetic tone below the level of injury and bradycardia, resulting from an unopposed vagal system. This hypotension worsens the ischemia present at the site of injury (see following). Moreover, there is a loss of au-toregulation following spinal cord injury.[21–23] The loss of autoregulation and unopposed vagal tone does not permit the body to compensate for the decrease in blood flow to the injured spinal cord.

Following spinal cord injury, there is an immediate de-crease in blood flow to the region of injury.[11,21,24–27] This decrease in flow persists for at least 24 hours. The cause of the local ischemia is not entirely clear. First there may be mechanical damage to small arteries and capillaries related to the primary injury. This injury may also induce vasospasm, which may also be induced or worsened by a vasoactive factor.[11] The vascular disruption may result in hemorrhage, especially in the gray matter of the spinal cord, thus worsening the ischemia.[28,29] Thrombosis of the capillaries[17,30] and veins[31–33] may also exacerbate the isch-emia. Thrombosis does not appear to affect the larger ar-teries such as the anterior spinal artery. Endothelial cell damage occurs. This may result in increased permeability, edema, and disruption of blood flow.[12,34–36] This dysfunc-tion has been demonstrated to occur as early as 1 to 2 hours following injury.[37] Glutamate and other neu-rotransmitters may also play a pivotal role in ischemia fol-lowing spinal cord injury.[12] Fracture displacement and fragments may also impinge on large and small spinal cord vessels. This may result in both local and global ischemic events.
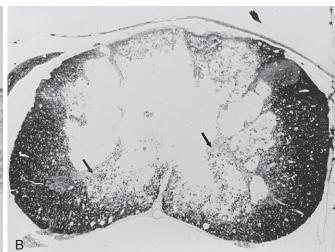
Fig. 2-5 Weeks following the acute injury, necrosis and cyst formation will be apparent. *A,* Numerous macrophages are present in the cysts. *B,* There is evidence of spared ventral and lateral white matter. *C,* Toluidine blue-stained thin plastic section. (Reprinted with permission from Rothman-Simeone. Herkowitz HN, Garfin SR, Balderston RA, Eismont FJ, et al. [eds]: The Spine, 4th ed. Philadelphia, Saunders, 1992.)

Following secondary central nervous system (CNS) injury there is a loss of intracellular energy substrates. The loss is caused by vascular damage and subsequent reperfusion-induced endothelium damage.[38] Mitochondrial dysfunction is a common event in cell injury in both ischemia and mechanical damage.[39] The anoxia leads to intracellular calcium accumulation, which irreversibly damages the mitochondria. This results in severe ATP depletion and eventual cell death.[40]

FREE RADICALS

Recent evidence has suggested that oxygen free radicals form early and are involved in the pathophysiology of CNS injury.[41] Markers of oxygen radical reactions have been demonstrated following injury. Examples include: increase in fatty acid oxidation products,[42] decrease in antioxidants[43,44] and the appearance of cholesterol oxidation products.[41,45]

Steroids inhibit lipid peroxidation to some extent[42] and may explain the neurologic improvement seen following experimental spinal cord injury.[45]

Lipid peroxidation begins almost immediately following experimental spinal cord injury.[12] This may be the reason that there is a time window for the administration of steroids following spinal cord injury, which has been demonstrated in humans to be less than 8 hours following acute spinal cord injury.[4]

INFLAMMATION

Immediately following traumatic spinal cord injury, the lesion site is infiltrated by neutrophils. These cells are able to phagocytize tissue debris and aid in homeostasis.[39] Their accumulation is significantly increased by 3 hours and remains elevated for up to 3 days.[46–48] These cells release proteases and reactive oxygen species. These elements have been shown to worsen the secondary injury.

Macrophages/monocytes are recruited and resident microglia are activated. This response increases over the first 7 days following injury and plateaus 2 to 4 weeks postinjury.[49] These cells are known to secrete a variety of cytotoxic substances that are part of the secondary injury process. These include tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), free radicals, cytokines, and nitrous oxide.[39] These cells are also actively involved with the phagocytosis of necrotic debris. The cytokines released are able to stimulate the recruitment of further inflammatory cells such as lymphocytes and induce the expression of cyclooxygenase, thus promoting the breakdown of arachidonic acid into substances such as prostaglandins that promote further tissue injury.[50,51] These substances promote increased vascular permeability as well as platelet aggregation and thrombosis.

Phospholipase A2 and C/diacylglycerol lipase pathways may also be activated. These pathways may also be activated by excitatory amino acids (see following). The activity of these phospholipases produces changes in membrane phospholipids composition, permeability, and fluidity, thus decreasing the integrity of plasma membranes.[52] This may result in cellular dysfunction, electrolyte accumulation, and/or cell death.

APOPTOSIS

Apoptosis, or programmed cell death occurs during embryonic development and in many disease states. There is a characteristic cellular phenotype, it is initiated by physiologic stimuli, and requires energy for new protein production.[53,54] Furthermore, an inflammatory response is not initiated with cell death. A family of cysteine proteins, the caspases, are thought to play a pivotal role in this process.[12] The caspase-3 apoptotic pathway is activated after experimental spinal cord injury. It may occur early in neurons at the site of injury and in a delayed fashion in oligodendroglia in the vicinity of the injury and at distant sites.[55] Excitatory amino acids, such as glutamate (see later) also have been suggested to contribute to apoptosis.

Apoptosis has recently been demonstrated in the human spinal cord.[56] It occurs at the site of the lesion as well as in ascending and descending white matter tracts, distant to the injury.[56] Oligodendrocytes are the main cell type that undergo apoptosis.[57,58] The mechanism has not been elucidated. It may occur because of changes in the cellular environment or because of Wallerian degeneration, or both.[59,60] Others have suggested that it may be the result of microglial activation or in response to activation of the FAS and p75 receptors.[61,62]

Substantial research is ongoing to explore the inhibition of these various pathways. The apoptotic pathway may be a good starting point because we have a growing knowledge of the caspase apoptotic pathway and the relatively slow time course lends itself to the development of treatment strategies. Agents that target this pathway may prevent neurons and oligodendrocytes from entering the pathway and offer neuroprotection following injury.

ELECTROLYTE DYSFUNCTION AND EXCITOTOXICITY

Excitatory neurotransmitters, glutamate and aspartate, accumulate following spinal cord injury.[63] The resulting increase in glutamate reaches neurotoxic levels. Receptors, NMDA and AMPA/kainite receptors, activated by glutamate, destroy neurons through prolonged excitatory synaptic transmission. Hypoxia, related to ischemic changes as described, makes cells more susceptible to glutamate toxicity.[64]

Glutamate activation results in an increase in intracellular sodium,[65] producing edema and intracellular acidosis. Intracellular calcium accumulation also occurs. This has been intimately related to traumatic neuronal cell death.[66] The increase in calcium activates a number of calcium-dependent proteases and lipases. These include calpains, phospholipase A2, lipoxygenase, and cyclooxygenase. Calpains are involved in the degradation of CNS structural proteins.[67] Phospholipase, lipooxygenase, and cyclooxygenase result in the conversion of arachidonic acid into certain thromboxanes, prostaglandins, and leukotrienes. These substances contribute to reduce blood flow by causing platelet aggregation and vasoconstriction.[68] They also contribute to the formation of lipid peroxidation and the production of free radicals, which contribute significantly to secondary injury.

Cyclooxygenase-2 (COX-2) has received recent attention as a contributor to the secondary injury following spinal cord injury. Its mRNA and protein expression have been demonstrated following experimental spinal cord injury.[69] Its induction may result in neuronal death by direct toxicity. Inhibition of COX-2 has been demonstrated to improve outcome following spinal cord injury in experimental animals.[69]

CONTINUED COMPRESSION

Continued compression of the spinal cord following injury also has been implicated in continued injury. The pathophysiology of the cause of continued injury has not been

elucidated, but most likely is from the aforementioned mechanism, such as ischemia, hemorrhage, lipid peroxidation, and so forth. A considerable amount of experimental studies evaluating the duration of compression and the results of decompression exist. Dimar et al.[70] examined the placement of various-sized spacers in the spinal canal of rats and the results of timing of decompression. The authors found that the time until decompression demonstrated that the motor scores were consistently better the shorter the duration of spacer placement over the 6-week recovery period. Shields et al.[71] demonstrated that spinal cord damage was directly proportional to the duration of spinal cord compression. The authors found that the therapeutic window prior to decompression was 6 and 12 hours in their 43% and 38% stenosis-spinal cord injury groups to maintain locomotor activity.

The aforementioned early decompressive experimental data have never shown equally in human clinical trials. There are a number of Class III studies demonstrating a benefit to both early and delayed surgery for spinal cord injury. Interestingly, even delayed decompression had shown effectively suggesting that compression of the cord is an important contributing cause of neurologic dysfunction. Although decompression is effective, a therapeutic window has not been defined.

Theoretically, spinal injury results in instability of the spinal column and can lead to further trauma and neurologic worsening. Ducker et al.[72] evaluated five pharmacologic agents to limit secondary injury in rhesus monkeys. They found however that immobilization was more important and significantly limited the severity of injury compared with nonimmobilized animals or those treated pharmacologically. They concluded that spinal immobilization improves outcomes and should be a standard in the evaluation of other therapeutic regimens.

The exact pathophysiologic mechanism is not elucidated but not unreasonable immobilization prevents displacement and further cord impaction. This has direct clinical implications as a major goal as stated by Rogers—is to "protect the neural tissues from further trauma" which is best initially achieved with proper immobilization and traction.

CONCLUSION

Clinical syndromes observed following spinal cord injury are determined by the level of injury and the anatomical tracts involved. Despite many years spent investigating spinal cord injury there is still no effective pharmacologic or surgical intervention. Significant advances recently have been made in the understanding of the pathophysiology of spinal cord injury. Future research may enable physicians to interfere with these complex cascades to afford some amelioration of symptoms.

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C H A P T E R

3

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Evaluation and Early Management of Spinal Injury in a Polytrauma Patient

INTRODUCTION

Appropriate initial management of a multiply-injured patient with possible spine trauma requires an integrated approach by several medical disciplines including emergency medicine, trauma surgery, orthopedic surgery, and neurosurgery. Vigilant maintenance of spinal precautions during a thorough evaluation of visceral, musculoskeletal, and neurologic injuries is essential to minimize the risk of further neurologic injury. It has been has estimated that there are more than 50,000 spine fractures in the United States per year.[1] Where fractures of the spine coincide with other significant injuries, the mechanism is typically a high-energy impact sustained from a motor vehicle collision or a fall from significant height.[2–4] Among the elderly, lower-energy trauma may result in multiple fractures of osteoporotic bone including upper cervical fractures, thoracic and lumbar compression fractures, pelvic fractures, distal radius fractures, and fractures of the proximal femur and humerus.[2,5] Other injuries often associated with spine trauma include pulmonary and cardiac contusion and head and solid organ injury. In combination, these injuries may lead to acute respiratory compromise or hemodynamic collapse; the resultant hypotension and hypoxemia can further exacerbate an existing spinal cord injury. Once a polytrauma patient is stabilized, a prolonged inpatient hospitalization, rehabilitation, and convalescence generally follows. The severity of the multisystem injury and the premorbid health status of the patient are the most determinative factors for the patient's risk of systemic complications including aspiration pneumonia, deep venous thrombosis, and skin and wound breakdown.

CLINICAL MANAGEMENT

Accepted protocols for the initial assessment, resuscitation, and stabilization of polytrauma patients have been established by the American College of Surgeons in the form of Advanced Trauma Life Support (ATLS).[6] The purpose of this protocol-driven approach is to provide a consistent and standardized approach that methodically identifies and prioritizes life-threatening injuries while maintaining precautions necessary to prevent further harm. This approach is crucial in the polytrauma context where patients present under exigent circumstances and are often unable to provide a complete medical history or localize painful injuries. Under these conditions, occult spine injuries may be missed due to an initial focus on acute respiratory or circulatory issues, or because a comprehensive neurologic examination is not possible.[7,8]

PRIMARY SURVEY

Initial assessment of polytrauma patients generally occurs in the field on the arrival of emergency medical technicians or other trained health care providers. The primary ATLS survey follows the mnemonic A-B-C, with "A" representing airway, "B" representing breathing, and "C" representing circulation.[6] In the context of evaluating for a spinal injury, each of these initial steps includes pertinent information to the diagnosis and management of potential spine fracture. All polytrauma patients are presumed to have a cervical spine injury until such injury is excluded, so appropriate precautions should be instituted and maintained throughout the primary survey. In adults, the cervical spine is immobilized in line with a rigid cervical orthosis or with sandbags and by taping or strapping the head to a spinal board.[3,9] Children have a disproportionately large head, and positioning them on a flat spine board leads to inadvertent cervical spine flexion. This flexed posture may result in further spinal cord compression.[3,9] Children should therefore be transported on a pediatric trauma board with a recessed head section, or the

child's upper torso should be elevated to restore neutral cervical spine sagittal plane alignment.

AIRWAY

Assessment of the patient's airway begins immediately and includes observation of the patient's spontaneous respiratory effort and ability to move air. The upper airway can be obstructed by blood: teeth: facial, mandibular, or laryngeal trauma. This condition requires emergency intervention by removing the mechanical obstruction or by establishing a patent airway through intubation or possibly tracheostomy. Cervical spine precautions should be strictly maintained, especially in the setting of head injury or facial injury where the rate of associated cervical spine fracture has been reported to be 7% to 24%.[10–13] In the absence of spontaneous respiratory effort, spinal cord or central nervous system injury should be suspected.[14]

BREATHING

Evaluation of the patient's breathing correlates with identifying major thoracic injuries including tension pneumothorax, open pneumothorax, flail chest, and massive hemothorax. Each of these conditions may rapidly lead to respiratory compromise and require specific, urgent intervention. Severe injuries to the chest wall should raise the clinical suspicion for significant or unstable thoracic spine fracture. Although fractures of the thoracic spine constitute only 10% to 20% of all spine fractures, most thoracic spine fractures occur as a result of high-energy trauma or polytrauma.[15] Because of the relatively narrow spinal canal in the upper thoracic spine, up to 60% of thoracic spine fractures result in complete spinal cord injury.[15] Injuries to the anterior chest wall and sternum are also relevant to the assessment of spinal stability. The sternum-rib-costotransverse articulation contributes significant stability to the thoracic spine by acting as a physiologic buttress or "fourth column."[16,17] Though one or two adjacent rib fractures have not been shown to affect thoracic spine stiffness, multiple rib fractures and sternal fracture as observed with flail chest, open pneumothorax, or massive hemothorax have been shown to compromise the stability of thoracic spine fractures.[18]

CIRCULATION

Assessment of the patient's circulation within the primary survey involves measurement of the patient's heart rate and blood pressure. Polytrauma patients are often hypotensive as a result of hemorrhage. Aggressive fluid resuscitation should be instituted at the accident scene, and the response of heart rate, blood pressure, and urine output should be closely monitored. When initial fluid resuscitation using crystalloid does not correct hypotension, the use of universal donor blood (group O, Rh negative) is effective in maximizing

oxygen carrying capacity. Early use of pressors is indicated in the setting of a spinal cord injury with associated neurogenic shock.

The secondary survey will rapidly identify the most common areas responsible for blood loss including the abdomen, pelvis, and thighs. Severe chest trauma with significant blood accumulation should be identified during the assessment of the patient's breathing capacity and oxygenation. Abdominal examination should include inspection for ecchymoses or abrasions from lap belt injury, palpation for peritoneal signs, a Focused Assessment with Sonography (FAST), or diagnostic peritoneal lavage (DPL). From the standpoint of spinal injuries, there is a significant correlation (10% to 33%) between flexion-distraction injuries of the thoracolumbar spine and intra-abdominal injuries.[19,20] Blunt abdominal aortic trauma has been reported in six cases associated with thoracolumbar spine fractures.[21]

Unstable pelvic injuries may result in substantial blood loss into the retroperitoneal space. Physical examination of patients with pelvic ring injuries may reveal scrotal or labial swelling, blood at the urethral meatus, and lacerations of the perineum, rectum, or vagina. Provocative maneuvers such as compressing the iliac wings medially or applying an anterior-posterior stress against the anterior superior iliac spine (ASIS) should be performed only once as the maneuver may lead to disruption of early clot formation. Initial control of pelvic hemorrhage may require emergent stabilization or angiographic embolization. A retrospective review of 18,644 trauma patients presenting to a single Level 1 trauma center showed that pelvic fracture was a strong independent risk factor for cervical spine injury, increasing the risk ninefold.[22]

The thigh is another large potential space for significant blood loss, and without appropriate immobilization, a closed femur fracture can rapidly result in four units of blood loss into the thigh.[23] Femur fractures are usually the result of high energy trauma and are therefore associated with multiple injuries including spinal fractures. In a large, multicenter retrospective review of 201 patients with femoral shaft fractures, 3.5% were found to have associated thoracic or lumbar spine fractures.[24] The association between fractures of the femoral diaphysis and spine fractures is especially important because the positioning and traction involved with insertion of a femoral intramedullary rod may exacerbate an unstable spinal injury. In one reported series, 57% of spine fractures occurring with femoral shaft fractures were undiagnosed at the time of admission and initial evaluation.[24]

Persistent hypotension with bradycardia should raise clinical suspicion for neurogenic shock secondary to spinal cord injury above T6. Disruption of sympathetic efferent innervation results in decreased peripheral vascular resistance and diminished cardiac output. Bradycardia defined as persistent heart rate less than 50 and hypotension with systolic blood pressure less than 90 mmHg may be treated with intravenous pressors and in more severe cases, cardiac pacing.[25,26] Neurogenic shock

requires early recognition and treatment to avoid prolonged hypoxemia and hypotension, which will exacerbate existing spinal cord injury.

SECONDARY SURVEY

Once the polytrauma patient is hemodynamically stabilized, the secondary survey consists of a complete clinical assessment including the spine from the occiput through the sacrum. When the patient is alert, cooperative, and not intoxicated and when painful distracting injuries are not present, areas of pain or tenderness on palpation may guide further radiographic evaluation. In other cases, as discussed previously, patterns of injury including more easily appreciated head, facial, chest, pelvic, or lower extremity trauma should increase clinical suspicion for coincident occult spine injury. Known injury at one spinal level should prompt radiographic evaluation of the entire spine to identify possible noncontiguous spine trauma. In a large series of pediatric patients with spine trauma presenting to a single regional trauma center, fractures were distributed fairly equally among the cervical, thoracic, and lumbar spine with 36% cervical, 34% thoracic, and 29% lumbar injuries.[3] Seven percent of children had multilevel noncontiguous involvement.[3]

Log rolling the polytrauma patient enables inspection and palpation of posterior bony prominences while maintaining appropriate spinal precautions. Once the anterior cervical soft tissues have been examined and the field collar is replaced by a rigid cervical collar, the patient should be log rolled and the posterior aspect of the spine should be visually inspected for abrasions, ecchymosis, and for rare open spinal fracture. The spinous processes along the entire vertebral column should then be palpated to identify areas of tenderness, malalignment, step-off, or irregular diastasis between adjacent spinous processes. If the patient is appropriately positioned at this time, examination for perianal sensation, rectal sphincter tone, and the bulbocavernosus reflex may be performed. It should be emphasized that although log rolling is an accepted component of spinal precautions, log rolling does not immobilize the spinal column.[27,28] When the necessary radiographic studies have been completed and the patient has been log rolled for posterior physical examination, the spine board should be removed.

Once the patient is log rolled back to supine position, a complete sensorimotor neurologic examination including reflexes should be performed and documented in detail. The American Spinal Injury Association (ASIA) neurologic classification of spinal cord injury and the ASIA impairment scale are useful tools in precisely mapping motor or sensory deficits and correlating these findings to a specific spinal cord level (Fig. 3-1), reproduced from the ASIA. Accurate initial documentation of motor or sensory impairment is essential because this becomes the baseline against which subsequent examinations are compared to assess for neurologic deterioration or improvement. Early attempts to obtain an accurate

neurologic examination may be hindered by patient intoxication, sedation, altered level of consciousness, or spinal shock. Spinal shock refers to transient reflex depression of the spinal cord function below the level of injury leading to 24 to 72 hours of paralysis, loss of sensation, hypotonia, and areflexia. The end of spinal shock is generally heralded by return of the bulbocavernosus reflex mediated by the S1, S2, and S3 nerve roots. Neurologic deficits that persist after the return of the bulbocavernosus reflex are unlikely to recover.

The use of high-dose methylprednisolone in the context of spinal cord injury continues to be highly controversial. Under current guidance, patients with spinal cord injury evaluated within the first three hours should receive a bolus of methylprednisolone at a dose of 30 mg/kg of body weight followed by an infusion of methylprednisolone at a rate of 5.4 mg/kg of body weight for 24 hours. If the patient is evaluated between 3 and 8 hours after injury, the infusion should be continued for a total of 48 hours.[29] Contraindications to steroid use include patients more than 8 hours after injury, patients with nerve root or cauda equine symptoms only, gunshot wounds, uncontrolled diabetes, pregnancy, age younger than 13 years, and those receiving maintenance steroids for other purposes. To minimize the risk of gastric ulcer, patients on a steroid regimen should be treated with a H2 blocker.
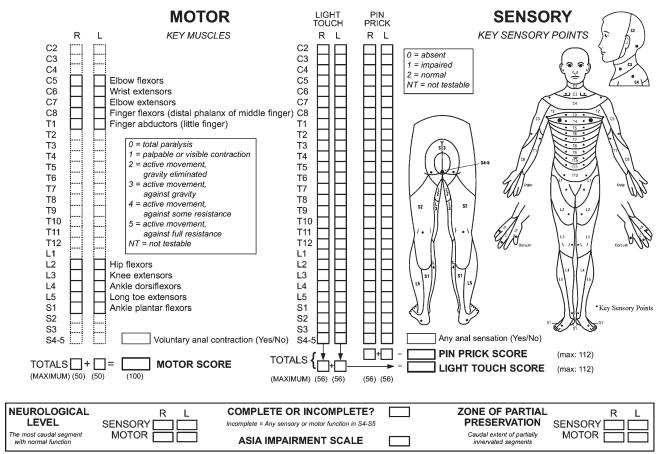
Putative evidence for the use of steroids is attributed to the National Acute Spinal Cord Injury Study (NASCIS), phases I, II, and III.[29–32] Although results from this series of large, prospective studies have been selectively interpreted to show a statistically significant improvement in motor and sensory function among patients receiving steroids compared with other agents or placebo,[29] others have challenged these conclusions.[33] Reviewing evidence from nine large studies including NASCIS I, II, and III, Hurlbert[33] concluded that routine use of methylprednisolone in patients with acute spinal cord injury was not supported and that prolonged steroid infusion may be harmful. Polytrauma patients may be especially susceptible to complications associated with the use of high-dose steroids including wound infections, pulmonary embolus, and sepsis.[30–32]

RADIOLOGIC ASSESSMENT

A standard radiographic survey of polytrauma patients includes anterior-posterior (AP) radiographs of the chest and pelvis. The AP chest radiograph may further delineate thoracic injuries identified during the primary survey, or it may detect more subtle findings including occult thoracic spine fracture. The chest radiograph should be studied to ensure that the spinous processes are evenly spaced, the vertebral heights are well-maintained, the pedicle spacing is regular, and that there is no anomalous coronal plane curvature or rotation of the thoracic spine. The AP pelvis radiograph is useful in assessing pelvic fractures and stability; it may also help predict blood loss from an unstable pelvic injury

Asia Impairment Scale

A = Complete: No motor or sensory function is preserved in the sacral segments S4-S5.

B = Incomplete: Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.

C = Incomplete: Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.

D = Incomplete: Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.

E = Normal: Motor and sensory function are normal.

Fig. 3-1 The American Spinal Injury Association (ASIA) neurologic classification of spinal cord injury and the ASIA impairment scale are useful tools in precisely mapping motor or sensory deficits and correlating these findings to a specific spinal cord level.

thereby guiding appropriate management.[34] Visible segments of the lumbar spine and the lumbosacral junction should also be studied on the AP pelvis, especially when lumbar spine fracture is suspected on the basis of history or clinical examination.

The traditional cervical spine radiograph series (consisting of AP, lateral, and open-mouth odontoid views) has been largely supplanted by computed tomography (CT) scans with sagittal and coronal plane reconstructions. This study is routinely obtained for polytrauma patients in most trauma centers. When a polytrauma patient is hemodynamically unstable, a technically well-performed lateral view of the cervical spine with visualization of the occipito-cervical region and cervicothoracic junction may be sufficient initial screening to enable the patient to be taken to the operating room as long as spinal precautions are continued and a rigid cervical orthosis is maintained.[35,36] Conversely, in the hemodynamically stable trauma patient, "pan scanning" (including head, neck, chest, abdomen, and pelvis) has become an accepted practice pattern and often obviates the initial chest and pelvic x-rays.

The ascendancy of CT imaging of the spine is attributable to several factors including improved institutional access to CT scanners, standardization of procedures for transporting polytrauma patients to obtain CT studies and the development of helical CT scan techniques enabling rapid acquisition of images. In the context of the multiply-injured patient with injuries resulting from high-velocity trauma, the frequency of head, thoracic, and abdominal injury generally necessitates head, chest, abdomen, and pelvis CT imaging, and further imaging of the cervical spine can be easily included. Use of CT to evaluate the cervical spine also avoids traditional difficulties of plain radiographs including inadequate visualization of the occipito-cervical and the cervicothoracic junctions, which have previously required additional studies. Even with technically adequate plain radiographs, sensitivity is only 52%,[37] and 15% to 61% of injuries may be missed.[38,39] In contrast, in a retrospective review of 3537 trauma patients, helical CT identified 99.3% of all fractures of the cervical, thoracic, and lumbar spine[40]; others have reported the sensitivity and specificity of cervical spine helical CT to be 98%.[37] Once a spine fracture is identified, CT images provide spatial characterization of the injured osseous structures, which is a valuable adjunct in preoperative planning.

Magnetic resonance imaging (MRI) is the most sensitive imaging method for evaluating soft tissue injury. Spine MRI provides excellent visualization of neural elements, ligaments, and intervertebral disks. Injury to soft-tissue structures alters their signal intensity, especially on T_2-weighted series, enabling precise localization of the compromised area. MRI can provide valuable information about spinal stability without the additional, albeit small, risk of spinal cord injury associated with physician directed flexion-extension radiographs.[41]

Despite these advantages, MRI may be difficult to obtain in polytrauma patients because of problems in transporting patients with ferromagnetic equipment including ventilators and monitors and because of the time required to perform a technically adequate scan. MRI may also be oversensitive. In a study of patients with upper cervical fractures, MRI did not change the treatment plan in neurologically intact patients.[42] Among patients with neurologic deficit however, MRI altered the preliminary treatment plan in one of four patients.[42] In general, MRI should be considered in patients with suspected ligamentous injury, in patients whose neurologic examination is not concordant with their findings by CT, and in patients with a progressive neurologic deficit.

MANAGEMENT OF THE POLYTRAUMA PATIENT

Appropriate management of the polytrauma patient requires close cooperation among multiple disciplines including general surgery, neurosurgery, orthopedic surgery, and critical care specialists. Where multiple operative interventions are necessary, the timing of surgery should be coordinated when feasible to minimize the number of anesthetic inductions and resultant anesthetic risk. The ideal timing of definitive orthopedic stabilization requires balancing of two opposing considerations. On one hand, the advantages of early stabilization of long-bone fractures include prevention of ongoing soft tissue trauma related to bony instability, earlier and less restrictive patient mobilization, and less postoperative morbidity.[43] On the other hand, the concept of "damage control orthopedics" originated from the appreciation that early, extensive surgery may exacerbate the systemic effects of polytrauma by prolonging the inflammatory response and by leading to higher circulating levels of proinflammatory cytokines including interleukin (IL)-1, tumor necrosis factor (TNF), and IL-6.[23] The systemic inflammatory syndrome triggered by polytrauma may lead to multiple organ failure, ARDS, and even death. To avoid these complications, authors have suggested that surgical treatment in the acute context should focus on control of hemorrhage, irrigation and debridement of wounds, and rapid skeletal stabilization.[23,44] These initial measures make possible a second stage of patient management focused on resuscitation and medical optimization that prepares the polytrauma patient for extended definitive surgical procedures. Established criteria for adequate resuscitation include: stable vital signs without the use of pharmacologic vasopressors, lactate level less than 2 mmol/L, international normalized ratio (INR) less than 1.25, and urine output greater than 1 ml/kg/hour.[23]

In the case of spinal injury, the appropriate timing of surgical decompression and stabilization remains controversial. Proposed benefits of early surgical intervention include rapid decompression of neural elements, prevention of further neurologic injury secondary to spinal instability, and facilitation of patient mobilization and transport once spinal precautions are

lifted. Data supporting some of these claims are equivocal. For example, although animal studies[45] and multiple retrospective reviews[46–50] suggest that early surgery has a positive effect on neurologic recovery, the only Level I evidence in this area did not show any difference in neurologic or functional outcomes among patients who had early surgery (<72 hours after spinal cord injury) compared with patients who had late surgery (>5 days after spinal cord injury).[51] In light of these contradictory reports, urgent decompression of acute cervical spinal cord injury, especially incomplete spinal cord lesions, remains a reasonable surgical option.[50,52]

When acute spinal cord decompression is not feasible as a result of concomitant injuries or clinical instability, provisional stabilization of known cervical spine injuries using a halo vest has been shown to be effective in preventing further neurologic injury.[53] In a retrospective review of 78 patients with known cervical spine injuries treated initially with halo vest stabilization, including 46 patients who had a subsequent surgical procedure, none had progressive neurologic deterioration after halo vest application.[53] Adherence to established safe procedures for halo vest application and pin site placement are essential to minimize morbidity associated with its use.[54,55]

In cases in which spinal cord compression is the result of unilateral or bilateral facet dislocation, early closed reduction using a halo ring or Gardner-Wells tongs should be attempted to reduce the traumatic deformity and re-establish the diameter of the spinal canal. With an awake, alert, cooperative patient, traction at an initial weight of 10 pounds may be started immediately. Once the initial weighted x-ray demonstrates a normal occipital-cervical junction, traction is then increased in 5- to 10-pound increments at 5- to 10-minute intervals. The patient should be re-evaluated clinically and radiographically before each increase in traction. The use of a c-arm or dedicated x-ray unit at the bedside is beneficial to expedite this treatment. Total traction is limited to 10 to 15 pounds per cervical spine level above the dislocation, or an increase in disk space height to more than 10 mm. Weighted traction above 70 to 80 pounds is rarely necessary to affect a reduction in the patient who has received adequate muscle relaxants. When the patient is obtunded or uncooperative or if a patient experiences neurologic deterioration during evaluation or attempted reduction, an MRI should be obtained to rule out associated disk protrusion.

From the perspective of damage control orthopedics, early definitive stabilization of spine fractures may function to facilitate resuscitation and care of the polytrauma patient in the intensive care setting without a significant increase in systemic morbidity. In a retrospective review of 291 patients with spinal fracture treated with either early (<72 hours after injury) or late (>72 hours after injury) fracture fixation, early fixation was associated with a lower incidence of hospital acquired pneumonia, a shorter intensive care unit stay, less ventilator dependence, and lower cost of hospitalization.[4,56] Even earlier spinal stabilization (<24 hours after injury) has been shown to be safe and effective in reducing complica-

tions including infection, pulmonary disease, and thromboembolism.[57] In cases in which spinal fracture coincides with lower extremity injuries including diaphyseal femur fractures as discussed previously, early spinal fracture fixation also reduces the likelihood of iatrogenic neurologic injury with the use of fracture-specific positioning or traction.

CONCLUSION

Polytrauma patients are assumed to have a spinal injury until a comprehensive clinical and radiologic evaluation can be completed. In the acute setting, respiratory, or hemodynamic instability may take precedence over the identification and treatment of spine injuries. During the primary and secondary assessment, spinal precautions should be strictly maintained. Patterns of injury, neck or back pain, and any neurologic deficit should alert the evaluating physician to a high probability of spine injury, and these findings should prompt a comprehensive radiologic evaluation. Once a spine injury is identified, the timing of surgery should reflect the principles of early decompression and damage control orthopedics.

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Physical Examination in Spinal Trauma: American Spinal Injury Association Examination and Spinal Cord Injury Syndromes

INTRODUCTION

Obtaining a complete history and performing a physical examination lead the physician to the correct diagnosis, establish the magnitude of the problem, and determine the appropriate treatment. In the case of trauma potentially affecting the spinal cord, an accurate account should be obtained from eyewitnesses to help determine the potential cause and mechanism of injury. An interpretation of the associated non-neural injuries may also provide clues to the type and direction of forces to which the spine has been subjected.[1]

The initial examination is important for establishing the level and extent of injury as a baseline for subsequent detection of secondary injuries to the spinal cord during the first few days of hospitalization.[2] For documentation purposes, an examination performed as soon as possible after injury may be important, but the initial examination often may be limited by concomitant brain injury, use of medication, and/or the influence of drug or alcohol intoxication.[2,3] Based on the results of multiple studies, the 72-hour examination is considered superior to the first-day examination for long-term prognostication.[2–9] Recently, Oleson et al.[10] reported that baseline lower-extremity pinprick preservation and sacral pinprick preservation at 4 weeks after injury have been associated with improved prognosis for ambulation.

The most accurate and useful assessment of spinal injury is one that is conducted with the use of a standard neurologic classification of spinal cord injury, such as defined by the American Spinal Injury Association (ASIA)[7,11,12] (Fig. 4-1). Evaluations of specific muscles and sensory functions are central elements of the examination. Examination of muscle stretch reflexes, including the bulbocavernosus reflex, is useful in assessing lesions of lower motor neurons.

In many instances, spinal shock is encountered at the time of the first posttraumatic evaluation. In such cases, upper motor neuron sensory loss and motor loss are associated with areflexia and flaccid paralysis below the level of injury. This is accompanied by atonic paralysis of the bladder, bowel, and stomach. Loss of sensation and impairment of autonomic function occur below the level of the lesion. This is postulated to occur secondary to sudden and abrupt interruption of descending excitatory influences. It lasts from 24 hours to 3 months, with an average duration of 3 weeks. The bulbocavernosus reflex often is the first reflex to return after spinal shock is over. It is tested in male patients by squeezing the glans penis and noting the contraction of the anal sphincter.[13,14] Minimal reflex activity usually is noted with the return of bulbocavernous reflex tested by perianal sensory stimulation and noting the contraction of the anal sphincter. If lower motor neuron injury is not present, reflex activity typically returns during the course of weeks or months.
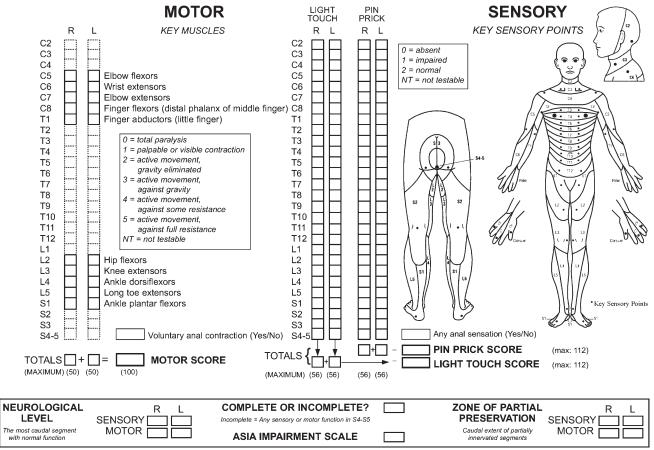
ASSIGNING AN ASIA LEVEL

To determine the ASIA level in a given case, one should refer to the ASIA form (Fig. 4-1) and conduct the following 10 steps:
1. Examine the 10 index muscles on both sides, five in the upper limb and five in the lower limb.
2. Examine the 28 dermatomes bilaterally for response to pinprick and light touch.
3. Complete a rectal examination for sensory and motor function assessment.
4. Determine left and right sensory levels.
5. Determine left and right motor levels.
6. Assign final motor and sensory levels.

Fig. 4-1 ASIA form. (Reprinted with permission from the American Spinal Injury Association: International Standards for Neurological Classification of Spinal Cord Injury, revised 2002, Chicago, IL.)

7. Calculate the motor and sensory scores.
8. Determine neurologic level.
9. Categorize injury as complete or incomplete based on the ASIA Impairment Scale (grades A through E).
10. Determine the zone of partial preservation.

EXAMINATIONS

MOTOR EXAMINATION

On the ASIA form, for each limb, the strength of five key muscles is recorded on a 6-point scale ranging from 0 to 5 (Fig. 4-2). The three components addressed while testing muscle strength are resistance to activity, role of gravity, and range of motion of the joint. The muscles should be examined in a rostral-to-caudal sequence with the patient in a supine position.[7] If muscle strength cannot be tested, it is reported as not testable (NT).

The score on each side is added (maximum = 50 points), and the points of both sides are combined to provide motor index scoring (maximum = 100 points). The motor score cannot be calculated if NT is documented. Optional elements of motor examination include testing the diaphragm (via fluoroscopy), deltoids, abdominals, medial hamstrings, and hip adductors.[11]

SENSORY EXAMINATION

Twenty-eight key dermatomes are tested for response to light touch and pinprick sensation on each side (Fig. 4-3). Zero, 1, or 2 points are awarded for absent sensation, impaired sensation, and normal sensation, respectively (see Fig. 4-3). The sensation of the face is used as a control measure for both light touch and pinprick. Inability to distinguish sharp pinprick sensation from dull sensation is recorded as absent sensation and is awarded 0 points on the pinprick scale.

MOTOR

KEY MUSCLES

R   L

C2
C3
C4
C5 — Elbow flexors
C6 — Wrist extensors
C7 — Elbow extensors
C8 — Finger flexors (distal phalanx of middle finger)
T1 — Finger abductors (little finger)
T2
T3
T4
T5
T6
T7
T8
T9
T10
T11
T12
L1
L2 — Hip flexors
L3 — Knee extensors
L4 — Ankle dorsiflexors
L5 — Long toe extensors
S1 — Ankle plantar flexors
S2
S3
S4-5

0 = total paralysis
1 = palpable or visible contraction
2 = active movement,
      gravity eliminated
3 = active movement,
      against gravity
4 = active movement,
      against some resistance
5 = active movement,
      against full resistance
NT = not testable

☐ Voluntary anal contraction (Yes/No)

TOTALS ☐ + ☐ = ☐ **MOTOR SCORE**
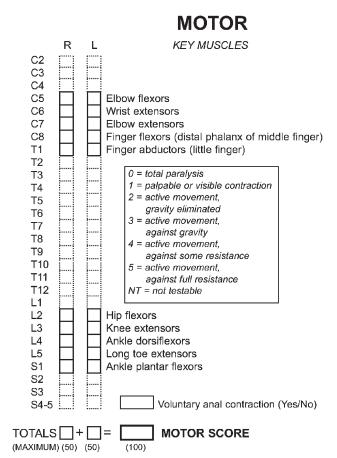(MAXIMUM) (50) (50)   (100)

Fig. 4-2 ASIA motor assessment of key muscles. (Reprinted with permission from the American Spinal Injury Association: International Standards for Neurological Classification of Spinal Cord Injury, revised 2002, Chicago, IL.)

Light touch   Pin prick
R L   R L

C2
C3
C4   0 = Absent
C5   1 = Impaired
C6   2 = Normal
C7   NT = Not testable
C8
T1
T2
T3
T4
T5
T6
T7
T8
T9
T10
T11
T12
L1
L2
L3
L4
L5
S1
S2
S3
S4–5   ☐ Any anal sensation (yes/no)

Totals { ☐ + ☐ = ☐ Pin prick score   (max: 112)
         ☐ + ☐ = ☐ Light touch score (max: 112)
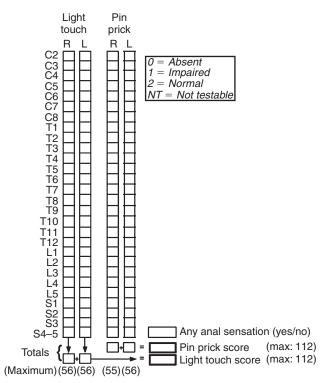(Maximum) (56)(56) (55)(56)

Fig. 4-3 Light touch and pinprick responses are scored for 28 dermatomes. (Reprinted with permission from the American Spinal Injury Association: International Standards for Neurological Classification of Spinal Cord Injury, revised 2002, Chicago, IL.)

Impaired sensation includes the ability to distinguish between sharp pinprick sensation and dull sensation, but the sensation is not as sharp as that felt on the face; it is awarded 1 point. Hyperesthesia to pinprick stimulation also is considered an impaired sensation and is awarded 1 point.[11]

A cotton applicator is used for assessment of light touch, and the swab should be moved over a distance of no more than 1 cm. If the sensation in a particular area is same as that on the face, 2 points are awarded. One point is awarded if the sensation is less than that felt on the face, and 0 points are awarded if light touch sensation is not appreciated at all. Most of the key points are tested on the anterior aspect of the body. Some key points are tested on the posterior aspects of the lower limbs, occiput, and shoulders, and on the dorsum of the digits of the hands. On the trunk, the key points are present in the midclavicular line.[11]

The sensory score is totaled for each side for both pinprick and light touch sensation (maximum combined score = 56 points for each sensation). The scores of both sides are then added to calculate a sensory scoring index (maximum combined score = 112 for pinprick and 112 for light touch sensation). If a sensation cannot be recorded for some reason, NT should be documented, in which case a sensory score cannot be calculated (Fig. 4-4).

RECTAL EXAMINATION

A rectal examination is performed to test voluntary anal contraction, which is documented as present or absent (Fig. 4-2). The examiner must be able to differentiate between voluntary contraction and reflex contraction of the anal sphincter. Deep anal sensation is also tested during this rectal examination and is recorded as present or absent (Fig. 4-3). The patient may have a feeling of pressure or touch and awareness of any other sense.[11]

LEVELS OF INJURY

SENSORY LEVEL

The sensory level of injury is the most caudal segment of the spinal segment that is associated with normal sensory function on both sides of the body, as assessed by response to pinprick and light touch (Fig. 4-3).

SENSORY
KEY SENSORY POINTS

0 = Absent
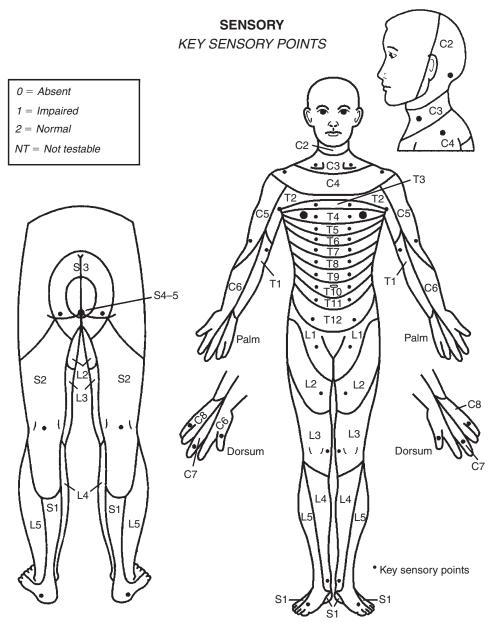*1 = Impaired*
2 = Normal
NT = Not testable

Fig. 4-4 Key sensory points are shown. (Reprinted with permission from the American Spinal Injury Association: International Standards for Neurological Classification of Spinal Cord Injury, revised 2002, Chicago, IL.)

MOTOR LEVEL

The motor level is the lowest normal motor segment, which may be different on each side of the body. It is defined as the lowest key muscle that has a grade of at least 3, providing the key muscles represented by segments above that level are graded as 5. The motor level is thus defined because most muscles are innervated by more than one nerve segment and a motor strength of 3 (antigravity) is considered to have intact innervation by the more rostral of the innervating segments.

NEUROLOGIC LEVEL

The neurologic level of injury is defined as the most caudal neurologic segment that retains normal sensory and motor function on both sides of the body (Fig. 4-5). Often, segments with normal function on one side of the body differ on the other side in terms of motor and sensory responsiveness. In such instances, the injury is best described by detailing the four segments (e.g., R-sensory, L-sensory, R-motor, L-motor).

NEUROLOGICAL LEVEL		R L	COMPLETE OR INCOMPLETE?		ZONE OF PARTIAL PRESERVATION		R L
The most caudal segment with normal function	SENSORY MOTOR	☐ ☐ ☐ ☐	*Incomplete = Any sensory or motor function in S4-S5* ASIA IMPAIRMENT SCALE	☐ ☐	*Caudal extent of partially innervated segments*	SENSORY MOTOR	☐ ☐ ☐ ☐

Fig. 4-5 Classification of spinal cord injury.

SKELETAL LEVEL

The skeletal level of injury is defined as the spinal level at which, by radiographic examination, the greatest vertebral damage is found. Of note, the neurologic level of injury often is different from the skeletal level of injury.

COMPLETE VERSUS INCOMPLETE INJURY

Careful rectal examination is required to determine whether the injury is complete or incomplete (Fig. 4-5). Complete spinal cord injury is indicated by complete absence of sensory and motor function in the lowest sacral segment.[8] If deep anal sensation or sensation at the anal mucocutaneous junction is reliably demonstrated or if voluntary control of the external anal sphincter is present, the lesion is considered incomplete. Sacral sparing is the presence of voluntary anal contraction and anal sensation.[2,7,11,14,15] Patients with incomplete injuries recover faster in the zones of injury than do those with complete injuries, but the degree of recovery is not necessarily greater.

ZONES OF PARTIAL PRESERVATION

The zone of partial preservation is present only with complete lesions and refers to myotomes and dermatomes caudal to the neurologic level of injury that remain partially innervated[11] (Fig. 4-5).

ASIA IMPAIRMENT SCALE

The ASIA Impairment Scale (Fig. 4-6) describes the degree of motor and sensory preservation below the level of injury and allows for description of the residual function of the injured spinal cord. Originally modified from the Frankel classification,[16] the ASIA Impairment Scale was created in 1992 and has been modified over the years. According to the scale, injuries are rated according to a spectrum ranging from complete functional impairment (grade A) to full motor and sensory preservation (grade E).

TETRAPLEGIA AND PARAPLEGIA

Tetraplegia can result from an injury to the spinal cord within the cervical canal, with damage to nerves of all four limbs, resulting in neurologic deficits in the upper extremities, trunk, lower extremities, and viscera. Cord transection

---

Asia Impairment Scale

A = **Complete:** No motor or sensory function is preserved in the sacral segments S4-S5.

B = **Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.

C = **Incomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.

D = **Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.

E = **Normal:** Motor and sensory function are normal.

Fig. 4-6 ASIA Impairment Scale defines grades A through E for assessing motor and sensory impairment. (Reprinted with permission from the American Spinal Injury Association: International Standards for Neurological Classification of Spinal Cord Injury, revised 2002, Chicago, IL.)

presents like features of bilateral hemisection after spinal shock, with flaccid paralysis. Patients with cord transection also have spastic bladder and loss of bowel function.[17,18]

Paraplegia results from spinal cord injury in the thoracic, lumbar, or sacral segments. The upper limb function is normal, but the trunk, lower extremities, and viscera experience neurologic deficits.

SPINAL CORD INJURY SYNDROMES

CENTRAL CORD SYNDROME

Central cord syndrome (Fig. 4-7) is the most common spinal cord injury syndrome and often is associated with neck hyperextension injuries in older persons with spondylosis. This is an incomplete injury characterized by predominant weakness of the upper limbs with relative sparing of the lower limb muscles. The pattern of neurologic deficit can be explained on the basis
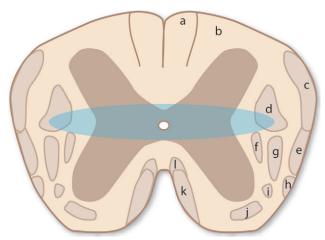
Fig. 4-7 Central cord syndrome. Medial nerve fibers of the lateral corticospinal tracts *(d)* are injured, leading to predominant weakness of the upper limbs and relative sparing of the lower limbs. *a,* Fasciculus gracilis; *b,* fasciculus cuneatus; *c,* posterior spinocerebellar tract; *d,* lateral corticospinal tract; *e,* anterior spinocerebellar tract; *f,* rubrospinal tract; *g,* lateral spinothalamic tract; *h,* spinoolivary tract; *i,* spinotectal tract; *j,* anterior spinothalamic tract; *k,* anterior corticospinal tract; *l,* medial longitudinal fasciculus.

Fig. 4-8 Brown-Séquard syndrome. Hemisection of the cord. *a,* Fasciculus gracilis; *b,* fasciculus cuneatus; *c,* posterior spinocerebellar tract; *d,* lateral corticospinal tract; *e,* anterior spinocerebellar tract; *f,* rubrospinal tract; *g,* lateral spinothalamic tract; *h,* spinoolivary tract; *i,* spinotectal tract; *j,* anterior spinothalamic tract; *k,* anterior corticospinal tract; *l,* medial longitudinal fasciculus.

of the somatotopic arrangement of the nerve fibers to upper and lower limb muscles in the corticospinal tract traversing the cervical cord. The nerve fibers to both upper limbs lie more centrally in the tract compared with the nerve fibers to the lower limbs. Sacral sensation is spared. Sensory loss is variable below the level of the lesion.[17–19]

BROWN-SÉQUARD SYNDROME

Brown-Séquard syndrome occurs in association with spinal cord hemisection (Fig. 4-8). Injury to the posterior white column leads to loss of discrimination touch, vibration, kinesthesia, and stereognosis on the same side, below the lesion. Lesion of the lateral spinothalamic tract leads to loss of temperature and pain sensation on the contralateral side, one to two segments below the level of the lesion.[17–20] The sensory tracts cross at different levels along the neural axis to the opposite side, which explains loss of temperature and pinprick sensation on the contralateral side and ipsilateral loss of touch and proprioception sensation (Fig. 4-9). Trauma to anterior white commissure leads to loss of bilateral pain and temperature sensation just at and one or two segments below the lesion. Flaccid paralysis occurs at the level of the lesion because of injury to anterior horn cells. With removal of all suprasegmental control, the alpha motor neurons driven by withdrawal afferents produce spinal defense reflexes of spinal automatism, with paralysis in flexion posture below the level of the lesion. This is because of injury to lateral corticospinal, lateral reticulospinal, medial reticulospinal, and vestibulospinal tracts.[18] Classical or "textbook"
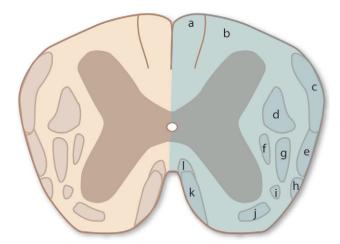
Brown-Séquard syndrome is rare, and patients often exhibit lesions with mixed characteristics.

ANTERIOR CORD SYNDROME

Anterior cord syndrome (Fig. 4-10) results from a lesion involving the anterior spinal artery or from direct trauma to the anterior two thirds of the spinal cord. The anterior spinal artery supplies the anterior two thirds of the spinal cord, and any lesions occurring anteriorly may interrupt the blood supply. This can happen with retropulsion of the disk or bone fragments and is sometimes associated with flexion injury of the cervical spine. Proprioception and light touch sensation are preserved because the posterior columns are intact. Loss of pain and temperature sensation and loss of motor function occur with loss of lateral spinothalamic and corticospinal tracts, respectively. Loss of bilateral pain and temperature occurs at the level of the lesion or one segment lower, with a lesion of anterior white commissure. Injury to the lateral corticospinal and lateral reticulospinal tract leads to spastic paralysis below the level of the lesion and may result in a spastic bladder and loss of bowel function. The patient presents with flaccid paralysis at the level of the injury because the anterior horn cells are damaged at that level.[2,14,17,18]

POSTERIOR CORD SYNDROME

Posterior cord syndrome is very rare and presents as loss of proprioception. Light touch and other sensations (pain and temperature) are preserved. Motor function is preserved to varying degrees.[17,18]
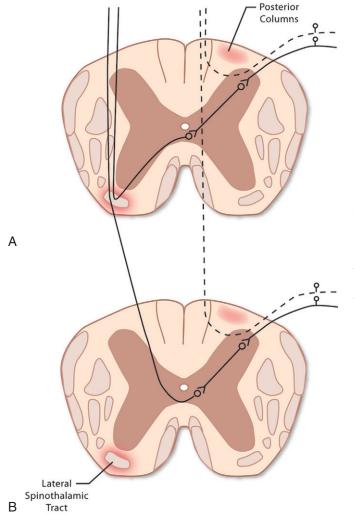
Fig. 4-9 Brown-Séquard syndrome. Pain and temperature sensation is lost on the contralateral side one or two levels below the lesion, and ipsilateral loss of touch and proprioception sensation has occurred. Pain and temperature sensation crosses to the contralateral side, to the lateral spinothalamic tract at the same level *(A)* or at one or two levels above *(B)*. Light touch and proprioception sensation ascends in the posterior columns without crossing in the spinal level.

CONUS MEDULLARIS SYNDROME

Conus medullaris syndrome (Fig. 4-11), caused by injuries to the tapering lower extremity of the spinal cord, often yields a combination of upper motor neuron and lower motor neuron symptoms and signs in the myotomes of the affected segments. This occurs because both the conus medullaris and the nerve roots may be involved at the lower level. Sensation loss, with sparing of touch sensation, occurs in a saddle distribution.[14] The patient presents with painless symmetric abnormalities. With high-level lesions (Fig. 4-11 level *A*), bulbocavernosus reflex and micturition reflex may be preserved, whereas with low-level lesions (Fig. 4-11 level *B*), the bladder, bowel, and lower limbs are areflexic. A lesion of the conus medullaris is characterized by devastating deficits in bowel and bladder function and a symmetrical sensory deficit in the saddle distribution. The deficits in lower extremity motor and sensory functions may be relatively minor if nerve roots L3 through S2 are not affected.[7,14]

CAUDA EQUINA SYNDROME

With cauda equina syndrome (Fig. 4-11 level *C*), the lumbar and sacral nerve roots are affected and the patient presents with lower motor neuron lesion, with flaccid paralysis and areflexic lower limbs. The bladder and bowel are areflexic, and male patients experience the loss of erectile function and the absence of the bulbocavernosus reflex. Sensory loss occurs in the root distribution, and pain may be present. The deficits associated with cauda equina syndrome can be asymmetrical.

SPINAL CORD INJURY IN CHILDREN

Injuries to the spinal cord and vertebral column are relatively uncommon in the age range of birth to 17 years. The pediatric incidence ranges from 1% to 10% of all spinal injuries. When evaluating spinal conditions in children, some characteristics need to be kept in mind.
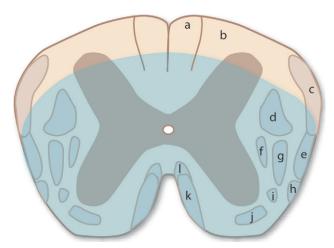
Fig. 4-10 Anterior cord syndrome. Loss of pain and temperature sensation and motor function occur with loss of lateral spinothalamic and corticospinal tracts, respectively. Proprioception and light touch sensation is preserved because the posterior columns are intact. *a*, Fasciculus gracilis; *b*, fasciculus cuneatus; *c*, posterior spinocerebellar tract; *d*, lateral corticospinal tract; *e*, anterior spinocerebellar tract; *f*, rubrospinal tract; *g*, lateral spinothalamic tract; *h*, spinoolivary tract; *i*, spinotectal tract; *j*, anterior spinothalamic tract; *k*, anterior corticospinal tract; *l*, medial longitudinal fasciculus.

Fig. 4-11 Conus medullaris (*A*, high; *B*, low) and cauda equina syndrome *(C)*. (Reprinted with permission from the American Spinal Injury Association: International Standards for Neurological Classification of Spinal Cord Injury, revised 2002, Chicago, IL.)

The spinal level and vertebral level reach the adult stage by 5 years of age. Up to the third month of fetal development, the spinal cord and vertebral column are at the same level. By birth, with differential growth, the spinal cord ends at the L2−L3 vertebrae, and by 5 years of age, the spinal cord ends at the L1−L2 level.[21]

Upper cervical vertebrae are injured more often in children younger than 8 years; when occurring in children older than 8 years, the pattern is similar to that observed in adults. Anatomic features of the cervical spine approach adult patterns between the ages of 8 and 10 years. Perhaps most important is the recognition that children can sustain significant spinal cord injuries without visible bony fractures or dislocations.[22–25] This spinal cord injury without radiographic abnormality (SCIWORA) accounts for 20% to 30% of all childhood spinal cord injuries. The predisposing factors are large head-to-neck ratio, elasticity of fibrocartilaginous spine, and horizontal orientation of the planes of the cervical facet joints.

Upper cervical SCIWORA has been reported to be associated with more severe neurologic consequences than lower cervical SCIWORA. Thoracic SCIWORA occurs less often. Spinal cord injury without radiographically shown lumbar spine abnormality is very rare.[24]

The paralysis may not present immediately after trauma and may present after a latent period of 30 minutes to 4 days.[1] This delay in diagnosis may occur secondary to the development of spinal cord ischemia or spinal instability.[24]
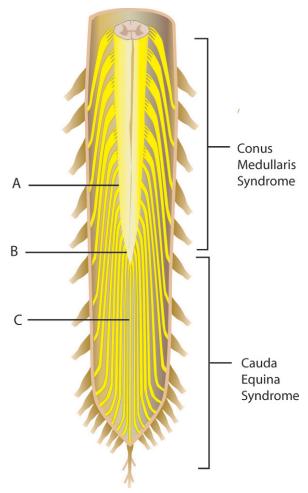
Pain in the cervical region should raise suspicion of cervical spine injury. Clinical manifestations of SCIWORA can range from subjective sensory abnormalities, such as tingling dysesthesia, numbness, ptosis, priapism, frank weakness, and paralysis, all without any visible radiographic abnormality.

Four distinct neurologic syndromes have been reported as occurring in patients with SCIWORA: complete cord transection (Frankel grade A),[1] central cord syndrome, partial cord syndrome, and Brown-Séquard syndrome.

CONSISTENCY OF EXAMINATION AND CLASSIFICATION

Much confusion regarding terminology and classification has been addressed by the standard neurologic classification of spinal cord injury. Consistent examination of patients with

spinal cord injury and consistent classification of the injuries have helped with research, documentation, treatment planning, rehabilitation, and prognosis.

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Radiographic Imaging of the Traumatically Injured Spine: Plain Radiographs, Computed Tomography, Magnetic Resonance Imaging, Angiography, Clearing the Cervical Spine in Trauma Patients

INTRODUCTION

Radiographic imaging of the spine in the trauma setting continues to evolve as technology improves. There is less reliance on plain radiography and greater use of advanced imaging in the initial assessment. However, plain radiographs continue to be an important tool in the characterization and treatment plans once an injury is identified. Plain radiographs also continue to be the predominant means of following the patient after injury.

Imaging is used in the identification, classification, and characterization of injuries. It guides the treatment plan and can be used to assess the effect of treatment. The energy and nature of the injury, level of consciousness, patient symptoms (e.g., pain, paresthesias, numbness), and objective findings on physical examination dictate the need for imaging and the type of imaging modalities that may be

necessary.[1–8] A subset of trauma patients who meet strict criteria do not require spine imaging. The criteria are lack of a high-risk mechanism, no pain or tenderness, no neurologic findings, no distracting injuries, and full ability to cooperate (no impairment of mental function from trauma, drugs, alcohol, or others causes). Although the need for palpation, range of motion, and lack of distracting injury is disputed in one study,[6] others place great significance on distracting injuries and note that any type of fracture should be considered a distracting injury.[9] The case reports that dispute this rule tend to have subtle clues to injury or do not strictly meet the previous criteria, such as having suffered loss of consciousness at the scene.[10–12] The principles used to determine the type of imaging required are the same regardless of the patient's age. However, interpretation can be more challenging in the pediatric population, particularly those children younger than 3 years because of the open growth plates and the lack of ability to cooperate with the examination. Anderson et al. developed a protocol that included radiographs and the NEXUS[13] criteria and were able to safely and efficiently clear the cervical spine in 507 pediatric patients with no late detection of injury.[14] Similarly, if the previous criteria are used to identify high-risk patients for thoracolumbar trauma, then a subset of asymptomatic patients, thoracolumbar patients, can avoid imaging studies.[15,16]

PLAIN RADIOGRAPHS

The classic initial imaging in an acute trauma patient includes a lateral cervical film at the same time the pelvis and chest films are taken. An anterior-posterior (AP) and open mouth odontoid views are added later if indicated based on injury mechanism, history, and physical findings. Thoracic and lumbar films are also added if the injury mechanism, history, and physical findings warrant further investigation. In the thoracic and lumbar spine an AP and lateral is required. If a single spine injury is identified then the entire length of the spine should be imaged because of a 1.6% to 23.8% rate of noncontiguous fractures.[17–21] The entire spine is also imaged if the patient is unconscious. The young pediatric patients who can cooperate with the history and physical warrant a more

thorough radiographic analysis based as well, taking into account the injury mechanism.

The standard cervical AP, lateral, and odontoid views have been reported to be as high as 92% sensitive in adults[22,23] and 94% in children,[24] but the increased use of CT scanning has been able to identify more injuries that had previously gone undetected.[25] Some of these injuries are minor and of little clinical significance but the ability to detect major fractures has also increased. In addition, the high sensitivity is assuming one can obtain adequate visualization of the entire length of the cervical spine. It is often difficult to adequately visualize the occipitocervical and cervicothoracic junctions (Figs. 5-1, *A* and *B*). To be considered adequate, the relationship of C7 on T1 must be visualized. With studies using computed tomography (CT) as a means of comparison for injury detection, the sensitivity of plain radiography for the cervical spine may be as low as 40% to 50%.[26] Attempts to improve the ability of plain radiography to detect injury has included a variety of special views such as lateral with arm traction, swimmer's view, trauma obliques,[27–29] and pillar views. Today less effort is put into obtaining a full set of cervical films as a means of screening the cervical spine because of the advent of helical CT scanners. Flexion extension cervical films still play an important role in clearance of the cervical spine and are discussed in more detail later in the chapter.

In assessing the cervical radiographs one should follow a systematic approach.[29,30] Once it is determined that the image of interest is that of the correct patient and the date is correct, then quality and adequacy are addressed. Next, the soft tissues shadows are reviewed to look for edema. The upper cervical spine is easier to assess and more reliable. Anterior to C3 should measure less than 3 to 5 mm and at C6 less than 15 mm. The lack of soft tissue swelling does not preclude the presence of a significant injury, but the presence of soft tissue swelling should act as clue to underlying injury. In subtle occipitocervical injuries, soft tissue swelling is common, and this area of the spine, in particular, should undergo close scrutiny if such a finding is noted (Figs. 5-2, *A-C*). The anterior soft tissue can falsely appear widened if the patient is intubated and is even more problematic in the pediatric population from crying. Next the anterior spinal line, the posterior spinal line, and the spinolaminar line should be followed (Fig. 5-3). The relationship between vertebrae at the spinous processes, disk, and facets should be evaluated for congruency, height, subluxation, translation, and distraction. Special attention should be given to the occipitocervical junction at this time. Lastly, the individual bony anatomy of each vertebra should be traced to find breaks in the cortical margins that represent fracture. Because the upper cervical vertebrae have unique anatomy and injury patterns, tracing the cortical margins and specifically looking for the typical fractures patterns is important. The pediatric cervical spine has unique features because of open growth plates and greater ligamentous laxity, and requires an understanding of developmental

A

B

Fig. 5-1 The shoulders may obstruct the view of the cervicothoracic joint, particularly in obese patients *(A)* making a CT necessary for clearance purposes. Adequate plain films must clearly visualize the occipitocervical and cervicothoracic junction *(B)*.
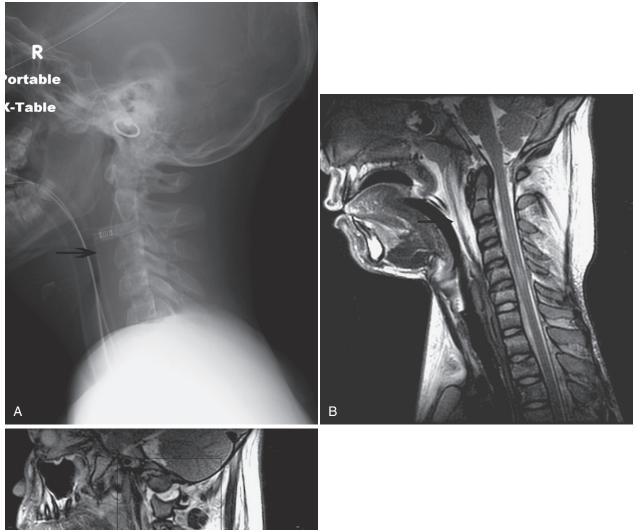
A

B

C

Fig. 5-2 Fifteen millimeters of anterior soft tissue swelling at the level of the C3 vertebral body as shown by the *arrow* in *A* should alert one to the possibility of subtle upper cervical pathology, particularly occipitocervical injury. The edema is easily seen in the sagittal T2 MRI *(B)* of the same patient as is the occipitocervical facet subluxation *(C).*

anatomy.[31,32] An understanding of the ossification centers of the atlas and axis can minimize confusion with fractures (Fig. 5-4).

In the thoracic and lumbar spine, AP and lateral views are the mainstay. A separate lateral of the upper thoracic spine may be required to penetrate the shoulders. Even with attempts to isolate the upper thoracic spine it is often poorly visualized and may require a CT scan, depending on index of suspicion. Because of these difficulties, careful inspection of the AP thoracic image is performed and can yield many clues to injury. Likewise, better definition of the lumbosacral junction is obtained with a spot lateral to
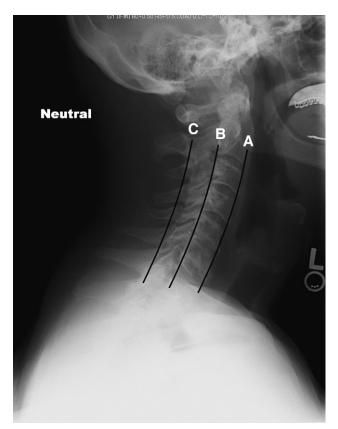
Fig. 5-3 A lateral cervical radiograph with the anterior cervical line *(A),* posterior cervical line *(B),* and spino-laminar line *(C).*

penetrate the pelvis. If an injury is identified at the thoracolumbar junction (which is typically at the upper or lower end of the film), then a separate AP and lateral should be repeated with the x-ray beam centered on the injured segment. These centered films will aid in assessing the fracture detail that may be important for treatment decisions. Similarly, upright or weight-bearing films can yield a substantial amount about the stability of certain fracture patterns and warrant discussion later in the book when treatment is reviewed because it has been reported to alter treatment decisions in up to 25% of patients[33] (Figs. 5-5, *A* and *B*). The lateral lumbar image often cuts off the caudal two thirds of the sacrum so if a sacral fracture is suspected, then a specific AP and lateral dedicated to the sacrum should be obtained.
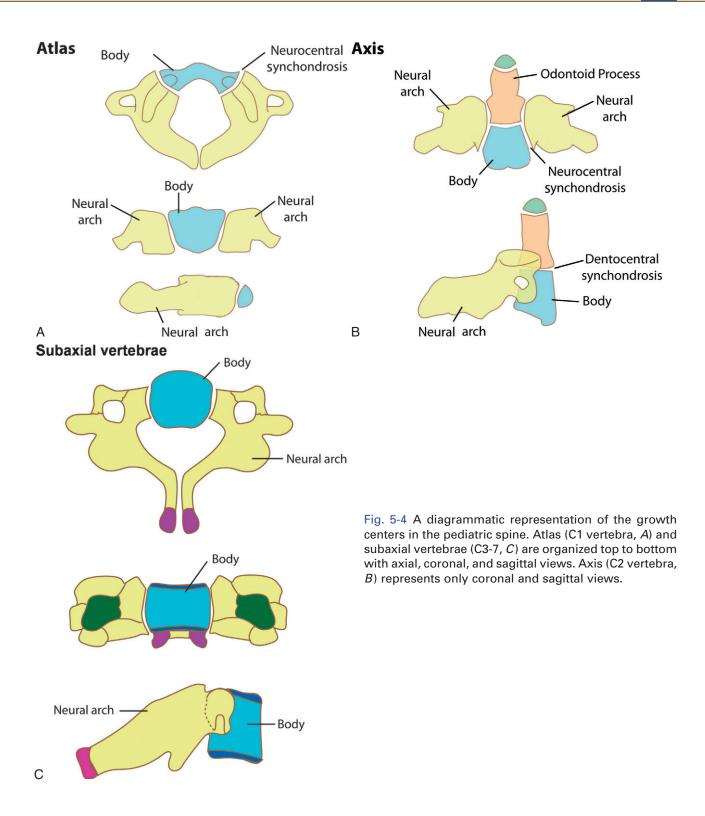
CT SCANNING

With the advent of helical scanning technology, the CT has taken on an even greater role in trauma evaluation. Once an injury is identified on plain radiographs, it is used to further characterize the amount of bony injury and a CT scan should be obtained in all patients in whom an injury has been identified on plain radiographs. Because

helical scanning data is acquired in a spiral fashion rather than slices, the sagittal and coronal reconstructions have a better resolution and portray the injury more accurately. For example, fractures that occur transversely, such as a fracture at the base of the odontoid or through the sacrum, were notoriously missed on older CT imaging because the fracture might be oriented perfectly inline with the axial slice of the CT making it invisible.[34] Even the reconstructions would miss such a fracture because the reconstructions were done from the axial slices[35] (Figs. 5-6, *A-C*). Although the axial images of a helical scan may not demonstrate this type of fracture well, it should be clearly visible on the reconstructions because the data are continuous. Because of this feature, as well as the speed of image acquisition,[36] the role of CT has expanded into one of clearance and has supplanted the need for plain films as a means of injury detection, at least in the high-energy trauma patient.[26,37–43] This appears to be true of the thoracolumbar spine and the cervical spine.[44] Interestingly, the use of plain radiographs and CT has almost reversed in that the CT is used to screen for injury; then a plain film is only obtained once the injury is identified as a baseline for subsequent follow-up. In the pediatric population in whom full visualization with plain films is typically easier, the use of CT for screening must be more carefully considered because it may not decrease the need for sedation or improve efficiency of evaluation, at the cost of increased irradiation.[45]

CT scans for use in the trauma setting should follow a preset protocol and this may vary somewhat between institutions but should follow some reasonable guidelines. Cervical CT scans should include the base of occiput to T4. The lower cervical spine and upper thoracic spine are poorly visualized on plain radiographs and are easily included with the cervical imaging (Figs. 5-7, *A-C*). The coronal reconstructions are most valuable for assessment of the upper cervical spine and thus should be aligned parallel to the odontoid. This alignment will best demonstrate injuries to the occipital-C2 region. The alignment of the axial imaging is more difficult to standardize. Ideally for the upper cervical spine it should be parallel to the C1 ring. However, this alignment may not be ideal for characterizing or identifying injuries in the subaxial spine because of the anatomical sagittal contours. For the sake of initial screening, the axial imaging is often oriented with the mid-cervical spine. If an injury is suspected or identified but poorly defined by such an orientation, the scan should be repeated with the gantry properly aligned with the segment of interest (Figs. 5-8, *A-D*). The orientation of the sagittal reconstructions is less challenging unless there is a coronal plane deformity.

Because plain radiographs are often adequate to screen the thoracic (except upper thoracic) and lumbar spine, CT is generally used to characterize injuries identified on those plain films. It may also be used in patients that continue to

Atlas

Axis

Subaxial vertebrae

A

B

C

Fig. 5-4 A diagrammatic representation of the growth centers in the pediatric spine. Atlas (C1 vertebra, *A*) and subaxial vertebrae (C3-7, *C*) are organized top to bottom with axial, coronal, and sagittal views. Axis (C2 vertebra, *B*) represents only coronal and sagittal views.

complain of pain, despite negative plain radiographs, to look for more subtle injury such as transverse process fractures. In high-energy trauma patients a helical CT of the chest, abdomen, and pelvis may be obtained for other purposes, which can then be used to screen the thoracic and lumbar spine.[46]

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) is used with more specific criteria than CT imaging and those criteria are continuing to evolve. Because the ability to visualize the soft tissues is better with MRI, it is used to assess for disk,
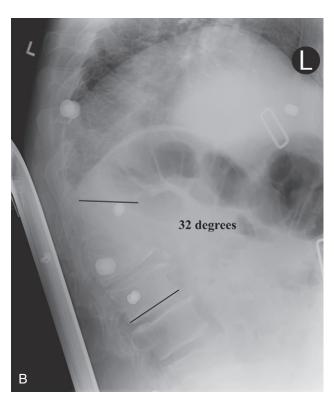
2 degrees

A

L

32 degrees

B

Fig. 5-5 Lateral supine lumbar radiograph with L1 burst fracture showing 2 degrees of kyphosis by Cobb measurement (A) and an increase in kyphosis to 32 degrees on standing in brace (B).

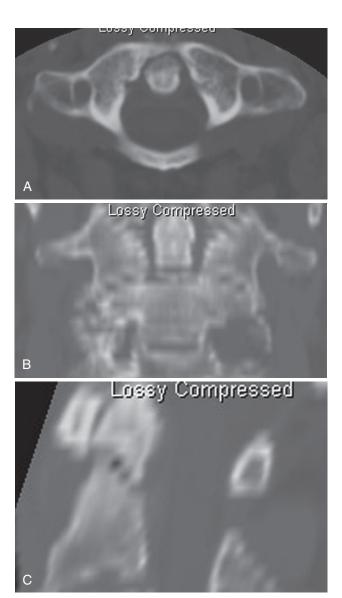Lossy Compressed

A

Lossy Compressed

B

Lossy Compressed

C

Fig. 5-6 An axial CT (A) can appear negative for fracture if the gantry is aligned parallel to the fracture plane, but reconstructions in the coronal and sagittal planes from a helical CT can demonstrate the fracture because the data is gathered in a spiral fashion (B and C).

ligamentous, and neurologic damage.[47] MRI plays a role as a screening study to clear the cervical spine as discussed later in the section on clearance and is also used when an injury has already been identified on plain radiography or CT studies.[48] The latter role is still more controversial in terms of deciding what injury patterns should be further studied with MRI and the timing of those studies. Benzel et al.[48a] studied MRI as the secondary screener after plain radiographs in 174 consecutive polytrauma patients with potential cervical injury, excluding those with injury identified on plain radiographs, and found 62 (36%) with treatable injury. This study was done in 1996 prior to

high-speed CT helical scanning that now is so rapid and high-definition that it would be hard to justify the time and difficulty in obtaining an MRI in all polytrauma patients. Others have proposed whole-body MRI to assess the entire spine for injury once one spine injury has been identified.[49,50] Some of this is best discussed when reviewing the particular injury and can be seen in later chapters. For the purpose of this chapter we can offer some generalities to indicate when an MRI may be beneficial or necessary.

Injuries to the upper cervical spine such as occipitocervical dissociations and atlantoaxial traumatic instability can be purely ligamentous injuries, making the MRI a valuable tool in identifying these injuries when suspected on initial studies. The ligamentous disruption may be seen more clearly and these injuries are often associated with a great deal of soft tissue swelling, which is also visualized well with MRI imaging (Fig. 5-9). Because routine cervical MRI protocols are not always designed with the occipital cervical junction[51,52] in mind, it may be worthwhile to

communicate directly with the radiologist that one is trying to assess ligamentous injury in this area so he or she can properly format the study to maximize the yield, generally involving a fat suppressed T2 imaging sequence. In the subaxial cervical spine, MRI is used to assess for disk disruption in facet dislocations, to rule out occult posterior ligamentous complex injury, and to evaluate the neurologic elements.[53] Assessment of disk herniation in association with facet dislocation is a topic in itself and is taken up in discussion of that injury in a later chapter. Determining occult ligamentous injury is covered in more detail later in this chapter in the section on spine clearance. However, it is also important to note that some seemingly benign bony injury patterns such as spinous process fractures can represent a more unstable injury if there is disruption of the posterior ligamentous complex in flexion or disruption of the anterior annulus in extension (Figs. 5-10, *A* and *B*). MRI may be able to distinguish these more significant injury patterns.
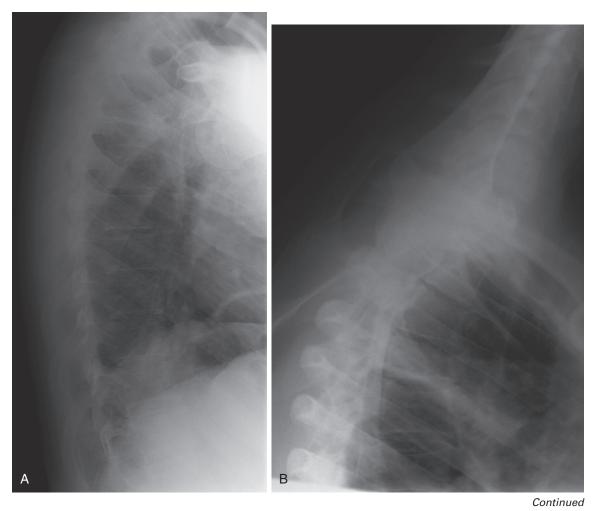

A

B

Continued

Fig. 5-7 The ability to visualize the upper thoracic spine is limited secondary to penetration of the shoulders *(A and B)* but any fractures are clearly seen via CT scanning *(C).*

Fig. 5-7, cont'd *C*

Lastly, MRI is warranted to evaluate damage to the neurologic elements. It can be used if there is a neurologic impairment that is not explained by the other imaging studies or does not correlate with a known level of injury.[54] In addition, there may be a role for MRI in better characterizing a spinal cord injury to determine prognosis and perhaps in the future guide interventions to aid in recovery. Several studies support the concept that hematoma within the cord, as well as the extent of that hematoma, is a very poor prognostic finding in the acute spinal cord injured patient.[55–60] This information may be particularly useful in the obtunded patient whose clinical neurologic examination is not available.

In the thoracic and lumbar spine, the role of MRI is similar in that it can be used to assess the neurologic injury and posterior ligamentous complex. It can also be used to assess for disk injury, but this use is controversial because the implications of such an injury are not well understood. It may be that the degree of disk injury predicts late pain and dysfunction but this is not known at the time of this writing and there are no widely held standards as to how this might affect treatment.[61,62] The most frequent use of MRI in the thoracolumbar region is in assessment of the posterior ligamentous complex.[63–66] At times, injury to the posterior ligamentous complex is clear from plain films or CT because of widening of the spinous processes, facet gaps, or rotational misalignments, but it can be more subtle. Injury to these structures may be the determining factor between an unstable and stable injury, thus having treatment implications.

When using MRI to assess the posterior ligamentous complex in the cervical or thoracolumbar spine, additional sagittal image sequences are used beyond the standard T1 and T2. The additional sequences are used to better assess for soft tissue edema and offer better contrast between the ligaments and surrounding structures. Generally,
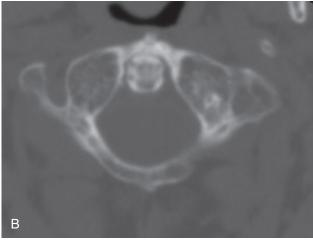

Fig. 5-8 *A,* The proper tilt of the CT gantry to allow visualization of the entire ring of C1 on one image. *B* and *C,* The alignment for the coronal reconstructions parallel to the odontoid process maximize imaging of the upper cervical spine *(D).*
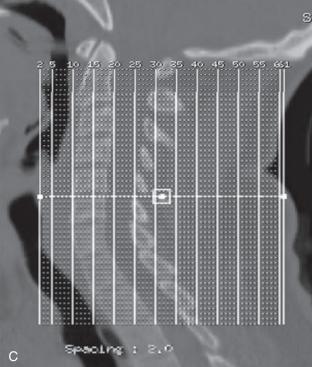
Fig. 5-8, cont'd *C* and *D*

Fig. 5-9 A sagittal T2-weighted MRI showing a pediatric occipitocervical dissociation with elevation of the tectorial membrane off the clivus, anterior subluxation of the skull on the atlas, and significant soft tissue edema.

some type of T2 fat suppression technique will best detect posterior ligamentous disruption.[67–70] The findings on these studies that most likely represent significant disruption is still being fully elucidated, but edema with the posterior ligaments, a clear discontinuity in either the "black stripe" at the posterior border of the canal or other structure such as the interspinous ligament, posterior longitudinal ligament, and supraspinous ligament have all been implicated. Of these, the disruption of the "black stripe" alone raises some questions[71] (Fig. 5-11).

Decisions regarding the need for MRI imaging in the pediatric population require more scrutiny because the patient must lie still for a substantial period of time, which may require anesthesia in the younger child. However, it is also a valuable tool in the assessment of spine trauma in this age group because they are often unable to cooperate with an examination, are vulnerable to spinal cord injury without radiographic abnormality (SCIWORA), and their bony anatomy may be difficult to interpret.

ANGIOGRAPHY

The improved technology of CT angiography (CTA) and magnetic resonance angiography (MRA) have made it easier to visualize the vascular structures about the spine in a less invasive manner that traditional angiography. MRA has been recommended by some[72] but is limited in its ability to detect spasm and small intimal tears.[73] Because patients are getting a CT of the injured segment, it is relatively simple to include
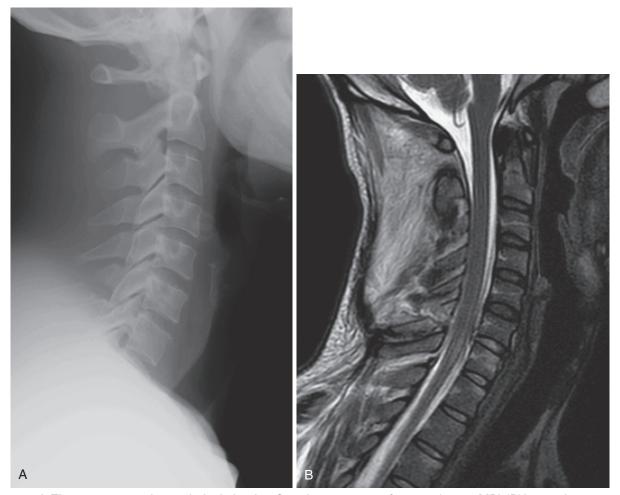
Fig. 5-10 *A,* There appears to be a relatively benign C6 spinous process fracture, but an MRI *(B)* better demonstrates the severity of this injury by revealing the soft tissue component, which includes disruption of the posterior annulus, edema in the C7 vertebral body, and spinal cord edema. All of these findings indicate that there was a more severe flexion component to the spine and it is likely unstable.
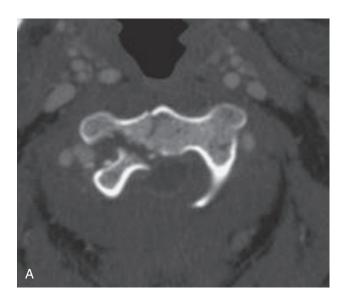
the necessary sequences for angiographic imaging. CTA has been reported to have sensitivity of 97.7%, specificity of 100%, positive predictive value of 99.3%, and negative predictive value of 99.3% in detecting occult vertebral artery injury.[74] As a result there has been greater interest in identifying vascular injury in association with spine trauma. This is most true of the vertebral artery because it traverses through the vertebral foramen, making it vulnerable to injury. As is often the case as imaging capability improves, our understanding of the meaning and implications of the findings lags behind. Almost every type of cervical fracture or dislocation has been implicated as a cause of vertebral artery injury, but subaxial facet fracture dislocations are reported most commonly.[75–80] We are now aware that injury to the vertebral artery is not an uncommon occurrence, with an estimated incidence between 17% and 44%,[81–85] but treatment may require anticoagulation, which has significant risk in the trauma patient, specifically one with a cervical fracture. In those

patients that demonstrate clinical features of vertebral artery injury, there is good agreement that radiographic evaluation of the artery is warranted. When no such clinical findings exist, it becomes more controversial because there is no agreement of what to do when an injury is identified in an asymptomatic patient[86] (Figs. 5-12, *A-C*).

CLEARING THE SPINE

Clearing the spine in trauma patients is a vital part of the initial assessment and has significant implications in long-term disability if an injury is missed. Thus, it must be done thoroughly and systematically to avoid such a catastrophe. It is the responsibility of all those involved in the care of these patients to ensure that this has been done properly. Adults and pediatric patients follow similar clearance protocols, although care must be taken in the very young child whose ability to fully cooperate is limited and SCIWORA[87] should be keep in

Fig. 5-11 A sagittal T2-weighted MRI image clearly showing disruption of the "black stripe" representing the posterior ligamentous complex *(arrow).*
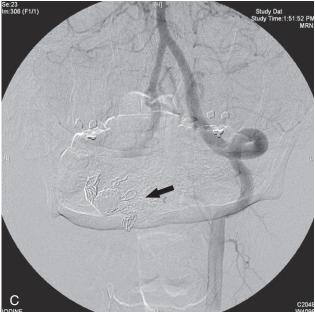
Fig. 5-12 *A,* There is a C2 fracture that extends into the vertebral foramen making it high risk for vertebral artery injury; thus, a vertebral arteriogram was obtained and demonstrates a vertebral artery injury with dye leakage *(arrow* in *B).* This patient was treated with an intra-arterial coiling *(arrow)* after a vertebral artery occlusion test, and retrograde flow through the Circle of Willis filling that vertebral artery is noted in *C.*
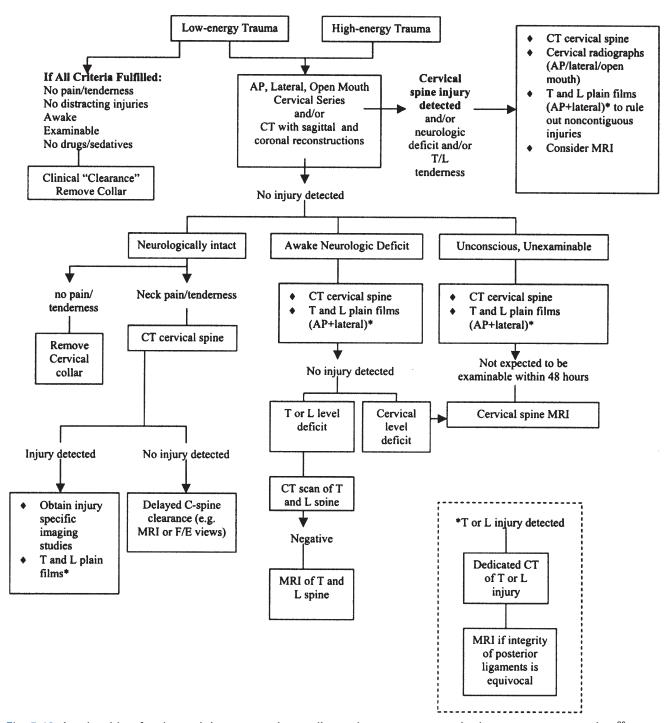
Fig. 5-13 An algorithm for determining appropriate radiographs as assessment in the acute trauma setting.[30]

mind for pediatric populations. Spine clearance lends itself well to protocols[30,49,88,89] (Fig. 5-13), and the following will offer a rationale to that process.

Some trauma patients can be cleared without the need for imaging but strict adherence to the criteria must be maintained and all other patients must include radiographic imaging for clearance. To meet criteria for immedi-

ate clearance, a patient must be fully alert and cooperative without alcohol or other medication that would alter the level of consciousness and have a low-risk mechanism of injury, no distracting injury, no pain, no tenderness, and no neurologic deficits per NEXUS criteria.[90] The NEXUS criteria also work in pediatric patients 8 yearfs of age and older.[13] Most of this is clear with the exception of defining

what constitutes a low-energy mechanism and a distracting injury. These two can be looked at together to clarify this issue. The extremes such as a patient with a high-speed roll-over motor vehicle accident and facial trauma has both a good mechanism and a clear distracting injury. On the other hand, a slip on ice and isolated external rotation fracture of the ankle without striking the head would constitute a low-energy mechanism and no distracting injury. Unfortunately, there is a large gray area. For example, the same patient in the last scenario suffers an ankle fracture but also strikes the head on the ice and has a large hematoma on the head but did not loss consciousness. Another example would be an elderly patient who suffers a fall from standing height in whom osteoporosis clearly makes the patient more susceptible to cervical fracture.[91] Bony fracture is most often cited as the distracter, which warrants radiographic spine imaging.[92,93] Chang et al.[9] reviewed 336 patients who underwent radiographic imaging solely on the basis of distracting injury and identified spinal fractures in 2.4% with bony fracture being the most common distracter. One clearly has to use judgment in the gray area but maintain a low threshold for radiographic evaluation because the consequences of a missed fracture outweigh the inconvenience of doing the workup.

Those patients that do not meet the previous criteria require radiographic clearance of the spine. Traditionally, cervical clearance began with a cross-table lateral in the trauma bay, later followed by open mouth odontoid and AP views. Trauma obliques and pillar views have been included in the past to better visualize the lateral mass complex, but these views have been largely supplanted by the helical CT scan.[37–42,44,46,94,95]

In high-energy trauma patients, particularly those going to the CT scanner for other reasons, the plain radiographs can be safely eliminated altogether if it is a helical scanner[26] as has been outlined in the section on CT imaging. Those patients that have neck pain despite negative adequate plain films, those with neurologic deficit, or those in whom the occipital cervical or cervical thoracic junctions are poorly visualized on plain radiographs (e.g., large or obese patients) will require a CT to complete their evaluation. A repeat lateral with traction on the arms to clear the shoulders or a swimmer's view can be use to expose the caudal levels but often these also fail to offer full visualization. Special circumstances such as ankylosing spondylitis in which visualization is often poor and the risk of fracture is high also are best suited for CT imaging.[96] If this group of patients is identified from the beginning, then again there is no need for plain radiographs and the evaluation can proceed to helical CT scan. This leaves a small subset of patients that are screened with plain films only. In some trauma institutes that follow this protocol, an initial cross-table lateral is still obtained in the trauma bay to rapidly screen for gross cervical deformity so that it is known prior to intubation and for moving the patient to the CT

scanner or other transports. If one is a purest, one could easily argue that this initial image only excludes a small portion of significant cervical trauma and that cervical precautions should be fully maintained until a complete assessment in completed eliminating the need even for the initial trauma lateral. Because CT scanners are readily available to most trauma bays, and given the rapidity that helical CT can be obtained, I am of the opinion that all initial screening plain films can be eliminated in the group of patients defined previously.

Because the entire cervical spine can usually be visualized in the pediatric population, helical CT scanning would be less often required,[45,97] perhaps with the exception of head-injured patients.[98] Also in patients that have a low suspicion for cervical trauma but fall into the gray area where they do not quite meet full criteria for clinical clearance alone, then plain radiographs are probably sufficient for clearance assuming the films are adequate (C7 on T1 is well visualized) and the patient has no pain, no tenderness, and no neurologic deficit that warrants a CT scan.[99]

For the thoracic and lumbar spine an AP and lateral plain films are generally sufficient to screen for significant trauma that threatens neurologic deterioration or dysfunction. If a patient continues to complain of pain despite negative plain films, then a CT scan may better identify subtle fractures, such as transverse process fractures, that would give the patient an explanation for the pain but usually do not carry a significant threat as an isolated injury. If the patient has undergone a helical CT of the chest, abdomen, and pelvis as part of the trauma workup, then this will usually be enough to screen the thoracic and lumbar spine. Sometimes only the abdomen and pelvis are scanned; thus, a thoracic AP and lateral would still be necessary. Because the upper thoracic spine is not well visualized on plain films, the cervical CT should include caudal levels to T4.

CT and plain radiographs are the studies of choice for bony injury, but an occult ligamentous injury could exist despite the fact that these studies are negative. Ligamentous injury can be implied from these studies based on misalignment between the bony structures. For example, widening of the spinous processes, gaps between facets, and rotation malignment would all imply disruption of the ligaments, but it may not be readily apparent (Figs. 5-14, *A-C*). This is one of the roles for MRI in the spine clearance process. It is more commonly necessary in the cervical spine than in the thoracolumbar spine.

Those patients who continue to complain of neck pain despite a negative CT scan require further evaluation before they can be cleared of ligamentous injury. There are two ways to accomplish this task. First, it can be done with dynamic flexion-extension lateral films. Just as in the initial screening studies, the lateral flexion and extension must visualize C7 on T1. This can be done under fluoroscopy with direct supervision to improve the yield and maximize safety, or it can be done as a one-shot flexion and one-shot extension with

patient in control with the endpoint being discomfort or the limit of motion. The latter is more common in the awake, alert patient that can control the excursion. If a patient is able to flex and extend through a full excursion defined as greater than 30 degrees from neutral and there is no evidence of instability, then the cervical spine can be cleared. Often the patient does not move through a full excursion and cannot be cleared, those patients should be maintained in a rigid collar immobilization and seen again in 1 to 2 weeks for repeat AP, odontoid, and lateral flexion-extension radiographs. If the repeat films are negative, then the neck can be cleared. Insko et al.[100] looked at 106 consecutive blunt trauma patients with flexion-extension views in the acute setting and found a false-negative rate of zero if the excursion was adequate. The excursion was inadequate in 30% of those patients, but they were still able to detect an injury in 4 (12.5%). Lewis et al.[101] reported one false negative in 141 patients but did not define that injury in their study. No neurologic injuries were noted in the papers reviewed with a combined number of 1518 patients.[100–103] It appears that

flexion-extension views can be useful in the acute setting because if an injury is identified, then the potential consequences of a delay in diagnosis are avoided, and the neck can be cleared if an adequate excursion is demonstrated without injury. Thus, only those patients with inadequate excursion and no injury identified are left for repeat follow-up. In the pediatric population, flexion-extension appears to be most useful when there are suspicious findings on the static plain radiographs.[104]

If a patient with negative CT imaging has neurologic complaints or objective neurologic findings, then an MRI would be warranted during the initial evaluation period (i.e., during that hospitalization or from the emergency room).[54] Some patients have new neurologic complaints when they return for 2-week follow-up and consideration should be given for an MRI. In the helmeted athlete, plain radiographs or CT can be obtained with helmet in place if necessary but must be removed prior to MRI scan.[105,106]

The final group of patients presents the most difficult challenge. This is the patient that is unable to cooperate

Fig. 5-14 CT scanning can be used to imply posterior ligamentous disruption with spinous process widening *(A)*, rotational malalignment *(B)*, and facet gapping *(arrow, C)*.
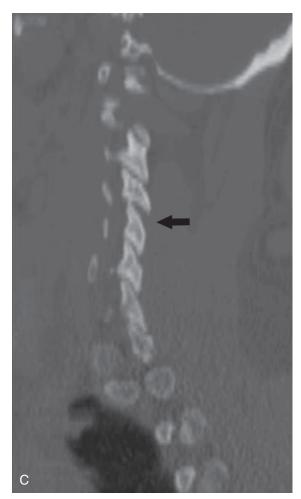
Fig. 5-14, cont'd *C*

the preceding paragraph for those patients that are cooperative from the beginning. If the mental status does not resolve, then other means of clearance need to be considered. The collar can be maintained initially but interferes with nursing care and can create decubiti, so clearance should be pursued with reasonable urgency.

In the obtunded patients, clearing the cervical spine of ligamentous injury can proceed with either flexion-extension laterals[108] or MRI.[109,110] There is some controversy in determining the most appropriate study, but each has advantages and disadvantages.[111]

If flexion-extension dynamic studies are to be used, they should be done under fluoroscopic control so the

because of an altered mental status. The altered mental status may be due to patient age (young[107] or old), patient intelligence level, closed head injury, intubation, recreation drugs or alcohol, or other drugs such as sedatives or narcotics given in the hospital. The initial evaluation would be the same with helical CT scan or traditional CT and plain films to assess for bony injury. If those are negative for acute trauma, then the potential for a pure ligamentous injury still exists (Figs. 5-15, *A-F*), the remainder of the evaluation is an attempt to prove that no occult ligamentous injury exists. If the cause of the altered mental status will potentially resolve within a few days then the patient can be maintained in a collar with cervical precautions, then assessed clinically for pain or neurologic findings after recovery. If they are now cooperative, deny pain, are nontender to palpation and axial load, have full range of motion without pain, and have no neurologic deficits, then they can be cleared without further study. If they are cooperative but do not meet those criteria, then further radiographic evaluation is necessary with flexion-extension or MRI as done in

Fig. 5-15 A 31-year-old man in a rollover motor vehicle accident who complains of neck pain and had some upper extremity paresthesias at the scene that have since resolved. *A-C* His sagittal trauma CT scan in the midline, right facet, and left facet, respectively, and was interpreted as normal. A flexion lateral in *D* clearly demonstrated an unstable subluxation at C4-C5. The disruption of the posterior ligamentous complex is seen on sagittal MRI views in the standard T2 and a fat suppressed T2 image *(E and F)*.
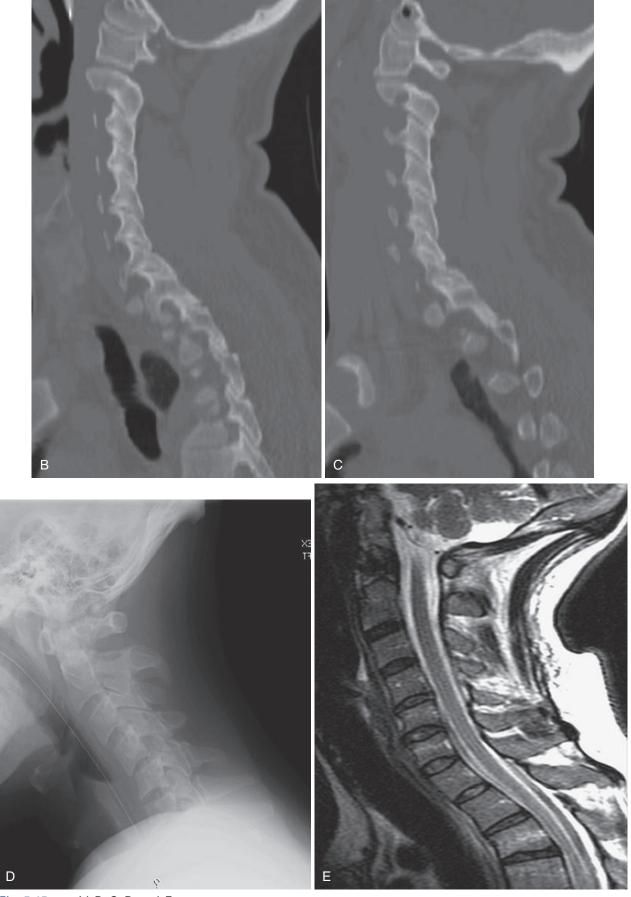
Continued

Fig. 5-15 cont'd *B*, *C*, *D*, and *E*

study can be stopped if there is any indication of instability to avoid harming the patient. Similar to plain radiographs, the cervicothoracic junction must be visualized, and in this group of patients who are often intubated and edematous it may be difficult.[112,113] A simple traction test prior to the flexion-extension may be adequate to demonstrate an instability, thus avoiding the more risky motions of the neck.[114] Because of the risks and difficult visibility with dynamic imaging, many centers prefer an MRI as the screening tool.[115]

When MRI used for this purpose, the scan should be done as soon after the injury as is medically reasonable to best demonstrate edema and minimize time in the collar. The study should include special sequences to show edema and to highlight the ligaments to better identify disruptions in those structures (the so called break in the black stripe). Obtaining an MRI presents its own set of logistical problems in transport from the intensive care unit, compatibility of the ventilator with MRI, and fitting the patient in the coil.

Schenarts et al.[116] looked at helical CT alone to assess the upper cervical spine and report that it detected all upper cervical injuries. Because the possibility of a significant, unstable cervical injury is rare in the face of a normal helical CT scan, some authors have postulated that the CT may be enough to clear the entire cervical spine in the

obtunded patient.[117–119] However, in the study by Hogan et al. there were 17 of 366 patients with injuries only identified on MRI. The ligamentous injuries were felt to involve only one column and thus stable, but several patients had various degrees of spinal cord injury detected. Based on the number of spinal cord injuries detected in this study, the remote possibility of undetected ligamentous injury and potential consequences of a missed injury, it is my opinion at this time that CT alone is inadequate for final clearance. Future study and continued improvement in CT capability may allow CT to be a definitive means of clearance in the near future.[120] The possibility of a significant ligamentous injury to the thoracolumbar in the face of a normal helical CT is remote enough; in fact no reported cases were identified that CT alone is adequate to clear the thoracolumbar spine, even in the obtunded patient. MRI is my preferred means of clearance under these circumstances in the adult and pediatric populations.[31]

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Timing of Surgery After Spinal Cord Injury

INTRODUCTION

In the United States, 10,000 to 12,000 cases of spinal cord injury occur annually. Currently, almost 300,000 people live with spinal cord injuries, many of whom are still young or middle-aged.[1–3] Direct costs involved with spinal cord injuries exceeded $7 billion in 1995. As medical and surgical methods improve, the number of survivors of spinal cord injuries will increase, as will the life spans of the survivors. Associated costs will therefore also increase.[4] It behooves us all to change the prognosis of this devastating injury.

Surgical and medical management of spinal cord injury remains controversial. This is because of the lack of Class I (Table 6-1) evidence regarding treatment and an incomplete understanding of the pathophysiology underlying spinal cord injuries.[3,5,6] Our current knowledge of spinal cord injuries divides them into primary and secondary injuries. The primary injury mechanism consists of a physical deformation and destruction of spinal cord tissue at the time of injury. This primary injury is thought to be untreatable. The primary injury mechanisms then lead to a cascade of events, resulting in continued destruction of the spinal cord. These events include vascular abnormalities, biochemical and electrolyte shifts, and cellular and molecular changes. Ischemia, loss of autoregulation, and hemorrhage are present after spinal cord injury. Calcium and sodium influx and increased extracellular concentration of potassium and excitatory neurotransmitters, such as glutamate, serotonin, and catecholamines, also occur. Cellular and molecular changes include infiltration of inflammatory cells, loss of mitochondrial potential, and activation of caspase proteins, leading to forms of apoptosis. Treatment with methylprednisolone is aimed at arresting several of the previously mentioned mechanisms. However, this treatment remains controversial. The National Acute Spinal Cord Injury Studies (NASCIS II and III) showed that methylprednisolone therapy might modestly improve neurologic outcomes if begun within 8 hours of injury.[7,8] NASCIS III suggests that treatment begun within 3 hours of injury needs to continue for only 24 hours—as opposed to 48 hours—when started 3 to 8 hours after injury. One important aspect of the NASCIS is that they are the very first randomized controlled trials to imply a potential therapeutic time window for spinal cord injuries. This partly validates the secondary injury hypothesis. This concept of a therapeutic time window is attractive not only for medical intervention but also for surgical treatment.[9–13]

CASE STUDY

An 18-year-old male patient was admitted to our institution after a motor vehicle collision that occurred in a remote region approximately 1000 km north of Toronto. At admission, the patient had incomplete injury at the C7 level. His admission American Spinal Injury Association (ASIA) grade was B and ASIA motor score was 46/100; he sustained no other injuries. Computed tomographic scans and magnetic resonance images obtained at admission are shown in Figures 6-1 and 6-2.[14,15] Methylprednisolone was intravenously administered at the time of admission. The patient was taken to the operating room, and his spinal cord was decompressed 48 hours after injury. Although a major effort was made to intervene urgently, the logistics of the long distances and remote location of the accident complicated the transfer. Twelve months later, the patient's ASIA grade had improved to C (Fig. 6-3). This case illustrates one of the most important unresolved questions in the operative management of spinal cord injuries: the timing of surgery to decompress the spinal cord. Would earlier intervention in this case (i.e., within 24 hours after injury) have resulted in increased recovery? Despite advancements in imaging, critical care, operative techniques, and instrumentation, it is still unknown whether early decompression of the spinal cord is advantageous and what time window after injury for decompression is associated with maximal benefit. In this chapter, we examine the controversy surrounding this topic and present the most recent data on the subject.

TABLE 6-1   Evidence Regarding Treatment

CLASS OF EVIDENCE	TYPE OF STUDY
Randomized controlled clinical trial	I
Prospective, nonrandomized studies	II
Retrospective studies, case series, expert opinion	III

ROLE OF NONSURGICAL MANAGEMENT ALONE IN SPINAL CORD INJURY

Considering the poor prognosis of spinal cord injury, it was previously thought that the best treatment was nonsurgical. This thought was bolstered by studies showing poorer outcomes in patients after laminectomies and a higher complication rate, including neurologic sequelae after surgery.[16–19] It is necessary to examine the outcomes of patients who are treated nonoperatively to carefully assess the positive or negative impact of surgical intervention. Frankel,[20] in his study of 612 conservatively managed patients, stated that 29% of the patients with Frankel Grade A improved at least one neurologic grade. Instability was noted in only four patients. Others also have shown neurologic improvement with conservative management. In contrast, Katoh et al.[21] reported that the conditions of 10% of patients with incomplete cervical cord injury deteriorated after nonsurgical treatment. However, all these studies constitute Class III evidence (see Table 6-1) because of their retrospective nature. Although conservative management remains a valid treatment option, especially for medically unstable patients, it is thought that with current neuroanesthesia, critical care, instrumentation, and surgical techniques, surgery will decrease morbidity and mortality through earlier spinal stabilization of the patient with spinal cord injury. This allows for earlier mobilization and easier respiratory care for the patient. Nonoperative management might still have merit for patients with central cord syndrome; however, with the modern advances in neuroanesthesia, critical care, and surgical techniques, nonoperative treatment of spinal cord injuries is no longer the preferred treatment option.[22]

EXPERIMENTAL STUDIES OF EARLY DECOMPRESSION

Several animal models have been used to show the potential benefits of early decompression.[23–25] Dimar et al.[24] showed improved neurologic recovery that was better with decreasing time of compression. They injured spinal cords in rats by using a weight drop and maintaining compression with epidural spacers. The compression was maintained for 0, 2, 6, 24, and 72 hours after weight drop. Carlson et al.[26] used a dog model to show greater neurologic recovery with earlier decompression. A hydraulic piston was used to produce spinal cord compression for 30 or 180 minutes. Somatosensory-evoked potentials and motor tests were conducted after the compression for up to 28 days. Dogs with only 30 minutes of compression were able to recover the evoked potentials with 1 hour of decompression, compared with no recovery in the 180-minute group. Similarly, the motor recovery was much improved in the early decompression group.

CLINICAL STUDIES OF EARLY DECOMPRESSION FOR SPINAL CORD INJURY

At present, no good Class I evidence shows that early decompression improves neurologic recovery. Several Class II studies have shown the feasibility of early decompression.[27–29]

Fig. 6-1 Computed tomographic sagittal reconstruction of the C6-C7 flexion distraction injury described in the case study. A 29% maximum canal compromise is present, as measured with both computed tomography and magnetic resonance imaging.[17,20]

Fig. 6-2 Sagittal view T2-weighted magnetic resonance image. Increased signal, indicative of possible hematoma formation, is seen in the spinal cord at the C6-C7 level.

Fig. 6-3 Mid-sagittal T2-weighted magnetic resonance image of the same patient shown in Figure 6-2, obtained after decompression and stabilization approximately 1 year after injury. A high-intensity signal, indicative of myelomalacia and cystic changes, can be seen at the C6 level.

A prospective nonrandomized case-controlled study of 208 patients by Tator et al.[30] showed that surgery resulted in a lower mortality rate than did conservative management despite an increased incidence of thromboembolic events. However, no difference in neurologic recovery between the groups could be discerned and the issue of timing of surgery was not examined. The concept of what constitutes early decompression remains controversial. It is uncertain whether any animal studies can be directly translatable, considering that clinical spinal cord injuries are so varied regarding mechanisms and force when compared with experimental models. Vaccaro et al.[31] reported, in a prospective randomized clinical trial, that early decompression (<72 hours) resulted in no difference in neurologic outcomes compared with delayed surgery (>5 days). Two main criticisms are applied to this study: (1) nearly one third of the patients were lost to follow-up and (2) some argue that 72 hours is too long a delay to be considered early decompression. This study should therefore be considered Class II evidence. Using less than 25 hours as the definition of early surgery and drawing from the NASCIS II database, Duh et al.[32] concluded that early decompression was beneficial compared with nonsurgical management, although no statistical significance was shown. Duh et al. also noted that surgery after 200 hours (delayed) might also improve outcomes. Another study of 91 patients with cervical spinal cord injuries showed that a greater percentage of patients who underwent early surgery (<10 hours) achieved better neurologic recovery than did the control group.[28] However, that study was a prospective nonrandomized trial, although the authors did show the achievability of early surgery in that all patients except one underwent surgery within 9 hours of arrival at the hospital. Another prospective nonrandomized study, conducted by Pointillart et al.,[33] could not find a difference in outcomes for patients who underwent surgery within 8 hours and those who underwent surgery after 8 eight hours. The study included 106 patients, 49 of whom underwent early surgery. The intent of that study, however, was to examine differences among methylprednisolone, nimodipine, and no medical treatment but not early surgery. In a retrospective study, Aebi et al.[34] reported that 75% of patients who recovered from spinal cord injuries underwent closed or open reduction within 6 hours.

Papadopoulos et al.[28] noted that patients who underwent closed reduction within 6 hours had better results than those who underwent open reduction within 12 hours. However, closed reduction is not without risks; in another study,[13] worsening of neurologic function occurred in 8.1% of 585 cases of closed reduction.

A meta-analysis of studies from 1966 to 2000, which focused on early versus late surgery, was undertaken by La Rosa et al.[35] The aims were to conclude whether neurologic recovery is enhanced when surgical decompression occurs within 24 hours, compared with late surgery or no surgery, in patients with traumatic spinal cord injuries and to quantify any

TABLE **6-2***   **Recommendations for Treatment**

LEVEL OF RECOMMENDATION	CLASS OF EVIDENCE	DETAILS
Standards	I	No standards regarding role and timing of decompression in acute spinal cord injury
Guidelines	II	Early surgery (<72 hr) can be safely performed in patients with spinal cord injuries if they are hemodynamically optimized Data support recommendation for urgent reduction of bilateral locked facets in patients with incomplete tetraplegia Data support recommendation for urgent decompression in patients with spinal cord injury experiencing neurologic deterioration
Options	III	Decompression is reasonable practice option for acute cervical spinal cord injuries; when possible (excluding patients with life-threatening multisystem trauma), it is recommended that urgent decompression be performed within 24 hr of spinal cord injuries Class III evidence suggests that early (<24 hr) surgery reduces length of stay for patients with acute spinal cord injuries and might reduce postinjury medical complications

*Reproduced with modifications from Fehlings M, Perrin RG: The timing of surgical intervention in the management of spinal cord injury: A systematic review of recent clinical evidence. Spine 31(suppl 11):S28−S35, 2006.

effect of early decompression. The study included 1687 patients who were categorized into early (within 24 hours), late (after 24 hours), and conservative (no surgery) groups. It was concluded that patients who underwent early surgery achieved better recovery than did those who underwent late surgery. However, because of the nature of the data, only results from patients with incomplete neurologic deficits would be reliable.

Class III evidence, mainly from retrospective studies, has suggested that late decompression might also be beneficial.[36] Duh et al.[32] noted a similar finding. In contrast, Waters et al.[29] reported that delayed surgery (after 14 days) did not improve outcomes. However, delayed surgery continues to be practiced for fear of the increased morbidity from early surgery that has been reported by various authors.[37–40]

Although solid evidence for early surgery to treat many types of traumatic spinal cord injuries remains elusive, sufficient evidence exists to support a recommendation of early reduction of bilateral locked facets in patients with incomplete injuries. A study of 76 patients, half of whom were admitted within 8 hours, showed that early reduction improved outcomes in patients with incomplete spinal cord injuries.[41] Other studies with class II evidence have presented similar findings.

Despite the evidence for bilateral facet dislocations, early decompression for improved neurologic outcomes remains unproven. Currently, the University of Toronto, Thomas Jefferson University, and the Spine Trauma Study Group are collaborating on a multicenter prospective trial to address that issue. The Surgical Timing in Acute Spinal Cord Injury Study (STASCIS) requires 450 patients and is currently open for enrollment.

CONCLUSION

Basic science studies have shown that the effects of trauma on spinal cord tissue are not instantaneous but require time to achieve maximal damage. This potential time window might explain why treatments such as methylprednisolone might have some effect on outcome. Animal studies also have shown that early decompression can have similar results. Whether these results from experimental laboratories can be directly translated to clinical practice remains to be determined. It is evident that early surgery is feasible and can be safely performed. However, because most evidence for early decompression remains class III, it can be considered only as a surgical option, not as a standard of care (Table 6-2).

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The Role of Pharmacologic Treatment of Patients with Acute Spinal Cord Injuries

INTRODUCTION

The permanent effects of traumatic spinal cord injury (SCI) result in irreversible alterations in motor, sensory, autonomic, and reflexive pathways.[1,2] The degree to which individuals are affected is dependent on the level and the extent of the lesion within the spinal cord. Neuronal loss and neurologic dysfunction have been attributed to physical and biochemical processes within the spinal cord following traumatic injury. Despite the significant impact of SCI on society, there has been little progress in developing an effective treatment for this disorder.

The pathologic sequelae of SCI, resulting in permanent neurologic deficits, are the product of two interrelated mechanisms; a primary and secondary injury. The primary injury results from the mechanical forces delivered to the spinal cord at the time of the trauma.[3] Tissue necrosis and loss of function are the consequence of neurons and glia being directly damaged by the physical deformation of the spinal cord.[4] Minimizing primary SCI could only be accomplished by primary prevention of the injury altogether. Following the primary injury, various complex pathophysiologic and biochemical reactions are activated, which have a detrimental effect on neural structures; these reactions are collectively referred to as the secondary injury.[1] Secondary injury develops within minutes to hours following the primary injury[5] and extends the region of damaged tissue far beyond the initial impact site. These time-dependent biochemical effects limit the potential of restorative processes, such as regeneration and reinnervation,[2] and ultimately exacerbate loss

of function. Reduction of secondary injury in spinal cord trauma may ameliorate tissue loss and functional decline after the initial insult. Understanding of the mechanisms and effects of secondary injury (as outlined in Chapter 2) is of critical importance to devise interventions that may ultimately improve the outcome of SCI patients. Although there has been considerable exciting and promising work published from the laboratory setting, this chapter focuses on the available pharmacologic treatments for acute SCI as they have been applied to the clinical arena. Three aspects of management are discussed: initial resuscitation, thromboembolic disease, and minimizing secondary injury.

RESUSCITATION

Hypotension in the setting of trauma is most commonly the result of systemic hypovolemia. However, in a patient with acute SCI, hypotension can also result from traumatic sympathectomy and loss of peripheral muscular and vasomotor tone. These manifestations are most common in complete injuries above T6. It has been well-recognized that systemic hypotension leads to worse outcomes in the setting of traumatic brain injury and, although not formally investigated in humans, animal data suggest the same processes are at play in SCI. Clinical data suggest that outcomes are superior if mean arterial pressure is maintained at or above 85 mmHg.[6–12] Although no specific trials have compared different treatment modalities, a general guideline has been proposed[12]: initial resuscitation should consist of intravenous crystalloid solutions; if hypotension persists then colloid solutions (blood, albumin or synthetic) should be added. If the patient is euvolemic and hypotensive, pharmacologic agents should be added; first a positive ionotrope, then an α-agonist.

THROMBOEMBOLISM

Deep venous thrombosis (DVT) and pulmonary embolism are common sequelae of SCI and a significant source of morbidity and mortality.[13,14] The true incidence is hard to extrapolate from the literature because of wide variations in treatment modalities and diagnostic tests used. Earlier data on those patients not receiving prophylaxis suggest an incidence as high as

100%.[15,16] However, despite the routine adoption of prophylactic measures, the incidence remains significantly elevated, in the range of 9% to 30%.[17–20]

There have been a number of clinical trials investigating various prophylactic treatment measures. Rotating beds[21] and low-dose heparin[22–28] have been commonly used with beneficial effect. Green and coworkers[29] published a lower incidence of DVT with a dose-adjusted heparin regimen compared with low-dose heparin, albeit with a higher rate of bleeding complications. Merli and colleagues[31] showed that the addition of external stimulating devices, either electrical[30] or mechanical, equally reduced the rate of DVT compared with low-dose heparin alone. The beneficial effects of pneumatic compression hose have been subsequently confirmed in a multivariate analysis of 428 spinal cord–injured patients.[32] Recently, the low molecular weight heparins have gained popularity for prophylaxis in this setting.[20,33,34] They appear to have an equal or superior protective effect and a lower risk of bleeding complications. A recent critical review[35] of the available literature suggests that prophylactic therapies are a standard of care in patients with severe neurologic impairment secondary to SCI. Low molecular weight heparin in combination with external compression devices and rotating beds may yield the greatest reduction in DVT incidence, but further study is required to confirm observation. Although the spinal cord injured population exhibits an increased risk of thromboembolic events for up to 1 year after injury,[36] most studies suggest that the incidence decreases after the first 3 months and the risk of prophylaxis may outweigh any benefit.[37–40] Therefore, it is suggested that patients be maintained on prophylaxis for the first 3 months after their injury.[14,35]

SECONDARY INJURY

A number of potential pharmacologic strategies have been investigated, targeting the reduction of the deleterious effects of secondary injury[41] and thereby minimizing the dysfunction associated with traumatic SCI. The following discusses medications used in past and current clinical trials.

METHYLPREDNISOLONE

Methylprednisolone (MP) has been extensively studied in both animal models and in humans and has the most widespread clinical use for traumatic SCI. However, its use has become a hotly debated topic in the past few years. MP is a highly lipophilic glucocorticoid that has been suggested to protect the spinal cord from secondary damage through many mechanisms. It has long been known that a high dose of MP (at least 30 mg/kg) is required in rats to achieve any beneficial effect on an injured spinal cord.[42] This dose far exceeds that which is necessary to activate corticosteroid receptors, which suggests that MP is working through mechanisms that are unrelated to its corticosteroid effect.[43] One

such mechanism that has been proposed is the antioxidant properties of MP, which protects the membrane lipids of neurons, axons, myelin, and various subcellular organelles, such as the mitochondria and nuclei.[44,45] MP is also felt to scavenge lipid peroxyl radicals, inhibit the lipid peroxidation cascade, and thereby reduce the severity of secondary damage.[46] These effects have been shown numerous times through the reduction of lipid peroxidation at various times after MP administration.[44,46–51] Although MP has been shown to improve various measurements of secondary damage experimentally, its effect on functional recovery has been controversial with many animal studies reporting no functional benefit with MP administration.[52,53] In addition, there have been problems associated with the use of high-dose MP, including hyperglycemia, infections, and acute corticosteroid myopathy.[54]

The potential for a human application of this treatment lead to the development of the National Acute Spinal Cord Injury Studies (NASCIS).[55–60] The initial NASCIS I[55,56] trial examined the use of 100 mg of MP versus 1000 mg of MP over 10 days; there was no placebo group. This was a randomized controlled trial and treatment started within 48 hours of injury. Outcomes were reported at 6 weeks and 6 months with no difference found between the groups. It should be noted that most patients were enrolled later in the 48-hour window and that the higher dose MP group had a higher incidence of wound infections. On further review of the animal literature, it was felt that the doses used were too low and adjusted for the subsequent trials. NASCIS II was a randomized, prospective, double-blinded, multicenter trial that was conducted from 1985 to 1988.[57,58] Patients received MP, naloxone, or placebo. All patients had to have an SCI and be randomized within 12 hours of injury. Patients randomized to MP received 30 mg/kg over 15 minutes, followed by a 45-minute pause and then a 23-hour infusion of 5.4 mg/kg/hour. The primary outcome results of the trial were negative; however, based on a subgroup post hoc analysis, this therapy was widely implemented in North America for patients with nonpenetrating injuries who could receive their first dose within 8 hours of their injury. In 1997, the NASCIS investigators reported on their third randomized trial. This study was conducted from 1991 to 1995.[59,60] Patients were treated with MP for 24 or 48 hours or with tirilazad mesylate. The MP group received the same dose as in NASCIS II and the tirilazad group was given 2.5 mg/kg every 6 hours for 48 hours. It should be noted that the tirilazad group also received the 30 mg/kg MP bolus initially. No placebo group was used as the investigators felt their previous trial provided strong enough evidence to warrant all patients receive MP. Based on their data (Fig. 7-1), the authors concluded that MP should be given for 48 hours after acute SCI, unless therapy is started within the first 3 hours, then treatment can be stopped after 24 hours. This regimen, again, was largely implemented; however, because of a higher incidence of adverse events, severe sepsis and pneumonia, in
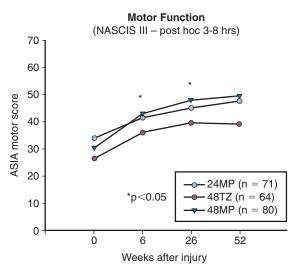
Fig. 7-1 Outcome data adapted from the NASCIS III[60] trial depicting ASIA motor scores as a function of time for the three treatment groups.

the 48-hour group several authors began to look at these trials more critically.[61–63]

There have been two other prospective trials[64,65] of MP in acute SCI and several retrospective studies.[66–68] With the exception of the Japanese study, for which there are some concerns regarding the randomization (Fig. 7-2),[64] these reports do not suggest a strong benefit to using MP in acute SCI. The NASCIS authors point out that their protocol was not adhered to in these studies, which could have had an adverse impact on the outcomes. Critics of the NASCIS studies question the significance of the clinical improvement,[61] the subgroup analysis,[61,62] the potential complications of the treatment,[62] and the differences within the various treatment groups.[63] The debate over the use of MP in acute SCI has led to the development of guidelines, that have
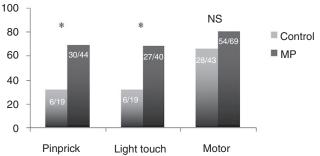

Fig. 7-2 Outcome data adapted from Otani et al.[64] illustrating improvement in sensory outcomes in patients treated with methylprednisolone. The percentage of patients that displayed motor improvement was not significantly different between groups.

been published by both the American[69] and Canadian[70] Neurosurgical Societies. These guidelines suggest that MP should be regarded as a "treatment option" and not a "standard of care" or even a "treatment guideline." However, given the contentious nature of this debate, readers are encouraged to review the available literature to formulate their own opinions so that therapy is based on scientific merit and not on peer pressure or fear of litigation.[71]

MONOSIALOTETRAHEXOSYLGANGLIOSIDE (GM-1 GANGLIOSIDE)

Gangliosides are complex glycolipids that are found in large quantities within neuronal cellular membranes. Experimental studies suggesting improved neuronal survival and neurite outgrowth led to the clinical Maryland GM-1 study.[72] This was a small (N = 34), randomized, blinded, placebo-controlled trial of patients with acute cervical or thoracic SCIs. Treatment (100 mg GM-1 daily) was initiated within the first 72 hours after injury and continued for 18 to 32 doses. After a follow-up period of 1 year, the treatment group showed a significantly greater improvement in function compared with the control group. This led to a larger randomized trial, the Sygen GM-1 Study.[73,74] This trial enrolled almost 800 patients from 1992 to 1997 from 28 different centers. Patients were randomized to receive placebo, low-dose, or high-dose GM-1. At the 26-week trial endpoint there was no significant difference between the groups; however, there was a trend for the high-dose group to have achieved their recovery faster. MP was given to all patients in both of these trials. In the first study 250 mg on admission followed by 125 mg every 6 hours for a total of 72 hours. In the second study, the much higher doses ascribed to NASCIS II were used in conjunction. This raises at least two possibilities for the loss of effect seen in the second trial: first, there is no true beneficial effect afforded by GM-1, or second, that an unknown, deleterious interaction with MP became more pronounced with the higher steroid dose. GM-1 is not currently being used for acute SCI.

THYROTROPIN-RELEASING HORMONE

Thyrotropin-releasing hormone (TRH) has been shown experimentally to have many beneficial effects in the setting of neurotrauma. With this support, a small trial of 20 patients was designed to assess the clinical effects.[75] Patients were randomized, in blinded fashion, to placebo or TRH (0.2 mg/kg bolus followed by 0.2 mg/kg/hour infusion for 6 hours). There were no adverse effects noted from the treatment. For incomplete injuries, there appeared to be a beneficial effect early in the treated group but no conclusions could be drawn at 1 year before patient dropout. Although the patient numbers were small, no differences in outcomes were appreciated in those with complete injuries.

NIMODIPINE

Nimodipine, a calcium channel antagonist, was investigated along with MP in a trial in France.[65] Patients in the MP arm received the NASCIS II regimen, whereas those in the nimodipine arm were treated with 0.015 mg/kg/hour for 2 hours, then 0.03 mg/kg/hour for 7 days. The two other arms were a combination therapy group and a placebo group. At 1 year, the investigators reported no difference between any of the groups. As the total number of patients in this study was only 106, it has been suggested that the conclusions could be susceptible to Type 2 error.[76]

CETHRIN

Cethrin is the recombinant antagonist to the protein Rho delivered along with a fibrin sealant that was developed by BioAxone Therapeutic Inc.[77] Rho is a small GTP-ase signaling protein that affects the response of neurons to growth inhibitory proteins. Furthermore, the Rho/Rho kinase pathway is upregulated following SCI, leading to an increase in apoptosis. The trial currently underway is a nonrandomized, uncontrolled Phase I/IIa trial for patients with acute complete cervical or thoracic SCI.[78] The drug is delivered at the time of surgical stabilization, which must be within 7 days of injury and applied directly to the dura overlying the injury site. The expected enrollment is 38 patients. Primary outcomes will be safety and pharmacokinetic data.

PROCORD

The poor penetration and reduced activity of macrophages within the central nervous system (CNS) following trauma is felt to contribute to the disparity seen in neuronal regeneration between the CNS and the peripheral nervous system (PNS). Procord is an experimental procedure devised by Proneuron Biotechnologies to address this deficiency.[79,80] The procedure requires "activation" of the patient's own macrophages by incubating them with a skin sample obtained from the patient's arm. The procedure is performed in a dedicated central laboratory with a turnaround time of less than 36 hours. The "activated macrophages" are then directly implanted into the spinal cord, below the area of injury, in six deposits. The current Phase II, randomized, open-label trial is enrolling ASIA A patients with traumatic injuries from C5-T11 in the United States and Israel.[81] The investigators plan to recruit 61 patients and randomize 2:1 in favor of treatment. All patients will require treatment within 14 days of injury and must have a visible lesion on a magnetic resonance imaging (MRI) scan. Rehabilitation will be standardized between the groups. The primary outcome, to be assessed at 1 year, is ASIA grade, with motor/sensory scores and bladder/bowel function as secondary outcomes.

MINOCYCLINE

Minocycline is a tetracycline derivative that has extensive clinical use in the treatment of chronic conditions such as acne, rosacea, and chronic periodontitis; has a good safety; and is well tolerated.[82,83] A number of experimental studies has shown evidence of its neuroprotective and antiapoptotic effects. In animal studies, minocycline has been shown to improve hind limb function and reduce tissue loss in the area of injury.[84] These data have been corroborated by other laboratories and have led to the initiation of a small, blinded, randomized, controlled pilot study at the University of Calgary in Alberta, Canada.

CONCLUSION

At this time, primary prevention is the best treatment for traumatic injuries to the spinal cord. The acute therapies available to us are directed at minimizing secondary injury and preventing complications from developing. Appropriate resuscitation reduces any ongoing ischemia, and prevention of thromboembolic disease reduces morbidity and mortality in the SCI population. Currently there are no widely accepted treatments for SCI with regard to managing the secondary injury. With numerous processes involved, and the complexity by which these processes influence and activate each other, it is difficult to establish the best and most effective mode of intervention. With further understanding of the biologic processes that occur after traumatic spinal cord, in terms of their exact mechanisms, their relationship with other processes, and their timeline of activation after injury, a reliable and efficient therapeutic strategy may be developed. Clinicians should be cautious in embracing data from animal models, or even clinical trials when the data are inconclusive or controversial. Premature adoption of "standards of care" may mandate inclusion of therapies in future trials, which may introduce bias or confounding variables that could weaken the experimental strength, and even outcomes, of these potential treatments. As clinicians and researchers, we need to address all aspects of acute SCI, such as pain, spasticity, and sexual and autonomic dysfunction, not just locomotion, as we work toward attaining the ultimate goal of functional recovery.

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C H A P T E R

8

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Intraoperative Neurophysiologic Monitoring of the Patient with Trauma

INTRODUCTION

Preservation of neurologic function is paramount when performing any spinal surgery, but it is of special concern in trauma cases in which acute neurologic injury and high degrees of spinal instability can place the neural elements at particular risk for injury. Previously, the Stagnara wake-up test served as the most reliable intraoperative monitoring method[1,2]; however, its use is limited. The inherent limitations of waking the patient include a time-consuming procedure, an inability to allow for repeated application, and a delay in diagnostic information, which make this form of intraoperative monitoring less than ideal.[3−6] In response to the demands for increased patient safety, intraoperative neuromonitoring techniques have been developed to be viable alternatives to the wake-up test.[5−16] The techniques to accurately detect changes in neurophysiologic function and to identify potential nerve injury have allowed the spine surgeon to avoid devastating clinical outcomes.

Very few studies identify the use of intraoperative neurophysiologic monitoring in traumatic spinal surgery.[5,8,11,17−19] The patient with multiple traumatic injuries presents a unique set of clinical characteristics and preoperative requirements that must be taken into account. The anesthetic requirements of a patient with polytrauma are uniquely dynamic and evolve secondary to extensive resuscitation and blood loss. The vacillating temperature shifts from prolonged exposure in the field, large-volume infusions, and operating room temperatures also significantly influence the findings and thresholds of the neuromonitoring tracings. A clear and

active line of communication must be maintained among the anesthesiologist, the surgeon, and the neuromonitoring technologist to interpret the intraoperative findings.

The purpose of this chapter is to outline the general principles of intraoperative neuromonitoring in spinal surgery. The latest techniques and procedures used in traumatic spinal surgery are emphasized. Several advancements and investigations in the recent literature have redefined the way neuromonitoring is applied and interpreted in cases of polytrauma. We attempt to provide a depth of detail necessary to form a solid foundation for understanding the intricacies and nuances unique to the patient with spine trauma.

TYPES OF MONITORING

SOMATOSENSORY EVOKED POTENTIALS

Somatosensory evoked potentials (SSEPs) are the electrical responses recorded over the scalp or spine after stimulation of the somatosensory (afferent) pathways located peripherally. They are indicators of the overall continuity of the intact somatosensory signaling pathway.[1,4,12−14,20−23] Intraoperative SSEP recordings can be obtained in either an invasive or noninvasive manner. Invasive spinal cord recording techniques, such as subarachnoid and epidural recording, have been reported in the literature.[20,21,24−33] These methods are more advantageous in that they produce a high-amplitude signal and have a higher resistance to anesthesia. Epidural recording electrodes can be introduced intraoperatively proximal to the operative field or percutaneously. Electrode placement in the subarachnoid or epidural space is theoretically most advantageous, but needle placement into the spinous processes or interspinous ligament usually provides adequate signal. Electrodes within the operative field also are an option but can interfere with surgery and might become inadvertently dislodged during the surgical procedure.

Noninvasive monitoring represents an equally effective means of yielding an SSEP waveform.[34] Transcutaneous sensors or subdermal needles are placed directly on the scalp, cervical spine, and/or peripherally to detect electrical somatosensory activity. Scalp needle electrodes are placed according to the International 10/20 System. With this designation, the

first letter corresponds to the anatomic location of the brain (frontal, central, parietal, occipital) coupled with a subscript letter "z" that refers to the midline (F_z, C_z, P_z, and O_z) or a subscript number (even numbers refer to the right cortical areas and odd numbers refer to the left).[12] For somatosensory monitoring, the designation of prime (C_z', C3′, and C4′) refers to the movement of the electrode 2 cm posterior from the standard position.[13,35] Application can be performed at the bedside with minimal discomfort to the patient and can even be placed before entering the surgical arena. Although subdermal needles have higher impedance, the overall ease of use and durability throughout the case make them a more advantageous option. Neither noninvasive nor invasive monitoring has proven to be superior in the setting of intraoperative neurologic injury.[30] The choice ultimately lies in patient characteristics and surgeon preference.

A standard SSEP montage is shown in Figure 8-1. An electrical stimulus is administered to the afferent cutaneous nerve. Any nerve can potentially be selected for stimulation with SSEPs. The upper extremity is monitored through the terminal branches of the median, radial, and ulnar nerves. The lower extremity usually is monitored through the posterior tibial or peroneal nerves. The median and posterior tibial nerves historically have the most described and characterized waveforms. Figure 8-1 shows a basic median nerve SSEP montage from an intraoperative recording. Five major peaks from the median nerve are identified: Erb's point, N_{13}, N_{18}, N_{20}, and P_{14}. The tracings are derived by placing the electrodes according to specific locations on the body, as outlined in Table 8-1. Erb's point serves as a means to calculate conduction velocity, show the presence of stimulation, and serve

as an indicator for preexisting peripheral nerve injury. "N" indicates a negative deflection and "P" a positive deflection, and the subscript number refers to the average latency in milliseconds. N_{13}, the potential derived from the C5 spinous process, is thought to originate from post-synaptic activity in the dorsal gray matter of the spinal cord. N_{13} is a subcortical potential that is largely unaffected by anesthetic agents and is therefore very useful intraoperatively. Two other potentials commonly are used: P_{14}, representing the caudal portion of the medial lemniscus; and N_{18}, originating from the upper brainstem and thalamus. N_{20} is a near-field potential that reflects activation of the primary cortical somatosensory receiving area. These cortical potentials are more sensitive to intraoperative conditions and anesthetic agents.

Lower limb stimulation is depicted in Figure 8-2. PF, LP, P_{31}, N_{34}, and P_{37} represent the five peak potentials derived from the posterior tibial nerve. Each peak has a corresponding function to those described for the median nerve. PF records the afferent volley and serves as the same reference purpose as Erb's point. LP is a far-field potential measured off the T12 level and iliac crest that often is very small and not commonly used intraoperatively. N_{34} is a far-field potential similar to the N_{18} median nerve, and P_{31} is most analogous to P_{14} after median nerve stimulation. P_{37} is a variable cortical potential often selected depending on the montage arrangement.

The size of the cortical SSEP from a single sensory stimulus is very small and difficult to record. Additionally, patient characteristics, electrical activity or "noise" from normal brain activity, and electronic instruments (e.g., Bovie electrocardiogram leads; Bovie Medical Corporation, St. Petersburg, FL)
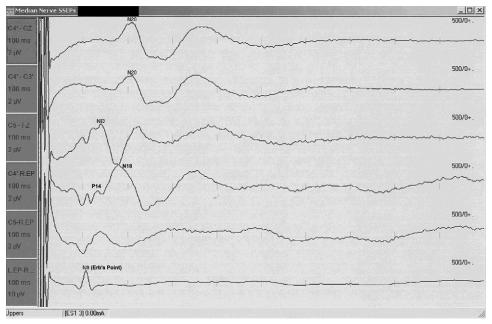

Fig. 8-1 Median nerve SSEP montage. Five major peaks are identified: Erb's point, N13, N18, N20, and P14. SSEP tracings are used to assess the overall integrity of the afferent pathways in the spinal cord.

TABLE **8-1**   Somatosensory Evoked Potentials and Their Associated Generators after Median Nerve Stimulation

NAME	LATENCY (ms)	PROPOSED GENERATOR
Erb's point	9	Trunks or divisions of brachial plexus/peripheral nerve
N_{13}	13	Spinal cord gray matter
P_{14}	14	Lower brainstem
N_{18}	18	Upper brainstem/thalamus
N_{20}	20	Somatosensory cortex
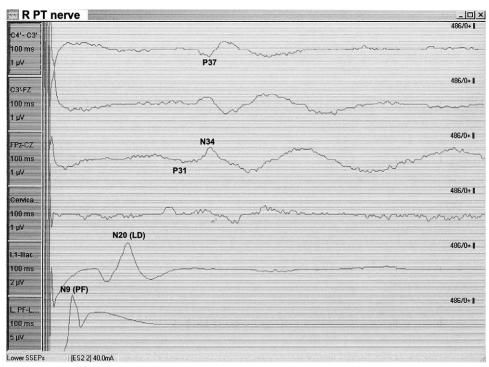
Fig. 8-2 Posterior tibial nerve SSEP montage. Five major peaks are identified: PF, LP, P31, N34, and P37. Lower extremity tracings can provide useful information to support upper extremity findings or can be used primarily for operations in the thoracolumbar spine.

require the filtering out of the various confounders.[36] The ability to identify SSEP cortical signals is aided by amplification and cumulative averaging of the desired signal. By taking the aggregate of 100 signal events, the signal-to-noise ratio improves by a factor of 10.[37] Sophisticated computer averaging and differential amplifiers help play a critical role in building a diagnostic SSEP waveform. As a result, the American EEG Society has set a standard for ideal technical specifications for obtaining routine evoked potentials in the operating room.[38]

Studies have reported numerous intraoperative factors that might alter SSEPs in addition to neurologic injury. These factors become increasingly important in the intraoperative setting to prevent false-positive outcomes. The most documented and correctable factors include hypothermia, hypotension, and inhalation agents. The effects of changing core body temperature by as little as 2 to 2.5°C have been shown to alter the latency and conduction velocity of all measured potentials.[39–42] The neuromonitoring technologist must adjust the thresholds for detecting neurologic injury. In a hypothermic patient, maintaining the standard 50% amplitude above baseline evoked potentials might not guarantee normal postoperative neural function.[43] A state of mild hypotension might be beneficial to limit blood loss during elective spinal surgery; however, in the trauma setting, especially with acute neurologic injury, induced hypotension has no role. Acute or prolonged hypotension might jeopardize spinal cord vascularity and cause spinal cord dysfunction with loss of evoked potential recordings.[25,44–46] Additionally, in animal studies, the effect of decreased perfusion and spinal deformation forces might be additive, further increasing the risk of exacerbating or inducing neurologic injury.[45,47] Inhalation agents, particularly volatile anesthetic agents that include isoflurane and its derivatives, have been shown to alter

or "blunt" somatic evoked potential firing.[27,48–52] Their mechanism of action is primarily on the suppression of sensory synaptic transmission.[53,54] Inhalational anesthetics might also induce hypotension,[55] which might explain a dual mechanism by which SSEPs are affected.

During the past decade, several attempts have been made to identify a threshold or cutoff that determines a significant change in SSEP to predict neurologic injury and allow the surgeon to take appropriate corrective action, if possible. The American Clinical Neurophysiology Society has defined criteria for abnormality as an absence of obligate waveforms and a prolongation of interpeak latency beyond 2.5 to 3 standard deviations above an appropriate population control.[56] Clinically, in a large multicenter study examining SSEP recordings during scoliosis surgery, a 50% decrement in amplitude or a 5% to 10% increase in peak latency from baseline was recommended as a threshold for abnormal SSEP change.[12–14] This has been supported not only by animal studies[57,58] but also by several other clinical trials.[20,21,59,60] Neurologic injury, however, is a dynamic process, and absolute thresholds often can be inadequate to predict persistent versus reversible neurologic injury. The immediate actions taken by the surgeon, the validity of the abnormal signals, and other factors play just as important a role in determining the predictive value of abnormal electrode recordings.

The clinical efficacy of SSEP monitoring has been studied extensively, although the bulk of available literature includes a mixture of diagnoses, including scoliosis and degenerative disease. Nuwer et al.[14] surveyed members of the Scoliosis Research Society regarding the morbidity and use of neuromonitoring. They reported that approximately half of the 97,000 cases used neuromonitoring—the vast majority in scoliosis cases. The rate of neurologic deficit, persistent deficits, and major neurologic deficits significantly decreased compared with previously reported historic values. The negative predictive value was 99.93%, which suggests that neuromonitoring was reliable if SSEPs remained stable. A low-positive predictive value of 42% reflected a tendency for false alarms; however, this value included those scenarios in which the surgeon might have averted a deficit because of a change in neuromonitoring. Interestingly, the cost-effectiveness of preventing one neurologic deficit for every 200 cases monitored was approximately $120,000. Epstein et al.[9] evaluated 100 consecutive cases of cervical surgery monitored with median and posterior tibial nerve SSEPs against an unmonitored historic control of 238 patients. The unmonitored group had a 3.7% incidence of quadriparesis, 70% good to excellent outcomes, and a 0.5% mortality rate. The monitored group fared better, with only one patient experiencing a temporary neurologic deficit, no mortalities occurring, and 85% good to excellent outcomes achieved. More recently, Khan et al.[61] reviewed 508 consecutive cases of cervical spine corpectomy surgery performed with intraoperative SSEP monitoring. The overall incidence of new postoperative neurologic deficit in this series of patients was 2.4% (11 with

nerve root injury and 1 with irreversible SSEP changes that led to permanent quadriplegia). The incidence of significant SSEP changes was 5.3% (27 of 508 patients). The calculated sensitivity and specificity of intraoperative SSEP monitoring for detecting intraoperative iatrogenic neurologic injury were 77.1% and 100%, respectively. If the isolated nerve root injuries were removed from the analysis, both the calculated sensitivity and the negative predictive values approached 100%.

MOTOR EVOKED POTENTIALS

Motor evoked potentials (MEPs) provide the surgeon with an alternative pathway of monitoring the integrity of the spinal cord. Whereas SSEPs are useful for dorsal column function, MEPs can accurately assess the integrity of the ventral portions of the spinal cord. MEP monitoring might be more sensitive to intraoperative changes in spinal cord function. The capacity to monitor both sensory and motor tracts of the spinal cord contributes to the overall completeness of spinal cord monitoring and ultimately increases the efficacy of preserving neurologic function.

ELECTRICAL STIMULATION AND TRANSCRANIAL MAGNETIC STIMULATION

Current MEP techniques essentially share a similar overall construct: antidromic activation of the motor pathways proximal to the surgical field with sensors recording responses distally at various levels. Stimulation is achieved either electrically or with transcranial magnetic stimulation (TMS). With transcranial electrical stimulation, a high-voltage, short-duration stimulus overcomes the high impedance of the scalp and induces a current that depolarizes neurons of the corticospinal tract.[62] More direct stimulation can be achieved with placement of the electrode on the spinal cord or through epidural electrodes. This technique is not often used in spinal surgery; however, these direct stimulation techniques produce potentials that permit rapid acquisition with strong responses. Distal recording can be obtained from either peripheral nerves or peripheral muscles as myogenic action potentials.

The disadvantages and relative contraindications to transcranial electrical stimulation must be considered and tailored to each surgical patient. The susceptibility of MEPs to anesthetic depression is well documented and must be carefully monitored throughout the entire procedure.[26,63–67] Pharmacologic muscular relaxation can even make MEP tracing impossible. Therefore, good communication with the anesthesia provider is essential before induction to avoid the use of long-acting agents, which might limit the ability to obtain reliable information from MEP recordings. For the patient with trauma, it often is desirable to obtain baseline recordings immediately on induction, before positioning. The anesthesiologist must take this factor into account when

planning medication selection. Transcranial electrical activation also has been contraindicated in patients with histories of seizure or abnormal electrical activity shown by electroencephalography.[68,69] Additionally, the use of transcranial electrical activation is discouraged for patients with skull fractures or implanted metallic intracranial devices because it could result in current shunting with excessively high local current densities.

TMS to achieve motor cortex stimulation and muscle action potentials was originally described by Barker et al.[70] In this construct, a pulsed magnetic field is passed across the cranium and generates an electrical current in underlying tissues, which leads to electrical activity in motor neurons. The main advantage of this technique is that it bypasses nociceptive afferents in the scalp and is better tolerated in the awake patient.[71] The disadvantages of this technique, however, might outweigh its use in the spinal surgery setting. TMS action potentials are less accurate, less reproducible,

and highly sensitive to anesthetic inhalational agents.[72,73] TMS also is contraindicated in patients with any intracranial metal implants, cochlear implants, cardiac implants, and pacemakers.

The standard MEP montage is shown in Figure 8-3. An electrical stimulus is generated in the motor cortex, which propagates down the corticospinal tract, activating anterior horn cells in the gray matter, traveling along peripheral nerves, and traversing the neuromuscular junction to activate a muscular contraction. Motor potentials, therefore, can be measured at various levels, including the epidural, peripheral nerve, and muscle levels. Recording compound motor action potentials (CMAPs) from muscle depolarization provides the most distal recording of motor evoked responses. CMAPs have large signals and do not need averaging to record their tracings. The key characteristics of a CMAP recording are in the amplitude of the positive deflection and the latency of the recording from stimulus. Neurogenic motor evoked
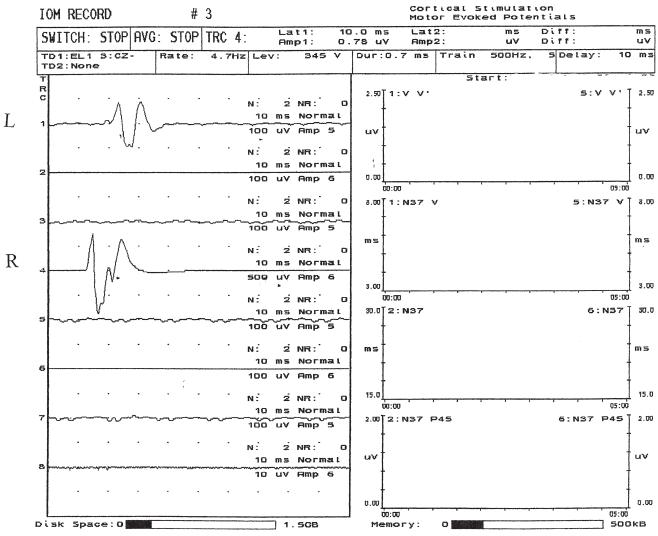

Fig. 8-3 Abductor pollicis brevis MEP montage. MEP tracings provide information regarding the integrity of the corticospinal tracts from an aggregation of multiple muscle action potentials.

potentials (NMEPs) recorded from peripheral nerves tend to be smaller in amplitude and therefore require averaging of several signals. NMEP recordings yield two waves: a direct (D) wave, which results from direct motor cortex stimulation and results in shorter latencies, and the indirect (I) waves, which are longer in latency, occur in series, and originate from synaptic depolarization "volleying" of the pyramidal tract neurons. The technical aspects of these waves lie beyond the scope of this chapter; however, the key to the NMEPs is their relative resistance to halogenated anesthetics.

The clinical efficacy of MEP as an adjunct to SSEP monitoring provides an invaluable source of information to the spine surgeon. SSEPs are mediated by the dorsal columns and therefore have no means of directly assessing ventral motor pathway function. SSEPs often can record a mixed nerve root with contributions from several sensory fibers from multiple nerve roots (e.g., posterior tibial nerve contributions from L4−S3). As a result, a monoradicular functional abnormality can be masked by the normal activity of the unaffected spinal nerve roots.[74−77] Several clinical studies and case reports have documented the presence of normal SSEPs despite clinically significant postoperative motor deficits.[17,18,31,32,46,78−85] These studies are important in defining the limitations of SSEP monitoring. However, the results should be evaluated critically and the usefulness of SSEP monitoring should not necessarily be discounted. It is arguable that these "false negative" outcomes are not truly false negatives but rather true positives in that they are correctly reporting the status of an intact spinal sensory column. It is important to understand that SSEPs might not be reliable as the sole monitoring modality of spinal cord integrity. If possible, a multimodal approach should be taken with MEPs for intraoperative monitoring of spinal cord function.

Monitoring techniques for MEPs were designed to overcome the inherent limitations of SSEPs. Owen et al.[86,87] showed increased sensitivity of MEPs over SSEPs in detecting cord integrity in animal studies. Inducing damaging effects of ischemia by overdistraction in a scoliosis model, they reported SSEPs to be insensitive to the effects of motor tract lesioning, and in cases of motor paraplegia, SSEPs remained unchanged despite abnormal MEPs.[77] Additionally, MEPs consistently degraded at lower levels of distraction than did SSEPs, indicating that a differential sensitivity of the motor and sensory tracts to ischemia exists.[57] Clinical studies examining the use of MEPs in spinal cord surgery have shown its effectiveness. Nagle et al.[88] presented the report of a cohort of 116 patients who underwent spinal operations ranging from deformity to spinal cord surgery with the use of both SSEP and MEP monitoring. MEPs could not be obtained at baseline for a higher percentage of patients compared with SSEPs. However, for all patients who had postoperative neurologic deficits, deterioration of evoked potentials was observed with MEPs. For one patient, normal SSEPs remained despite the abnormal loss of MEP signal. In 3.5% of cases, changes in the monitored signals led to major alterations in

the surgery. Pelosi et al.[18] further explored the importance of MEP signal changes in a cohort of 126 patients. SSEP and MEP monitoring was achieved in a majority of the patients with various spinal disorders that included deformity, trauma, infection, and degenerative and ankylosing spondylitis. Intraoperatively, 16 patients experienced evoked potential changes: one with only SSEP changes, seven with both SSEP and MEP changes, and eight with only MEP changes. Based on the results, the authors concluded that MEP changes were more predictive of postoperative neurologic complications. Specifically, "transient" MEP changes during instrumentation without SSEP changes probably were of no clinical significance. "Persistent" alterations of MEPs during instrumentation, regardless of SSEP changes, invariably led to postoperative motor deficits. Based on these studies, the importance of following MEP changes throughout surgery and altering surgical instrumentation and manipulation to correct for changes in MEP is clear.

ELECTROMYOGRAPHY

During pedicle screw instrumentation, potential injury to the neural elements is significant. The exiting nerve root sits within a few millimeters of the medial pedicle wall. Electromyography is an important tool for the surgeon during instrumentation of a traumatic spine. Spontaneous or stimulated muscle activity can be used to monitor mechanical injury to nerve roots related to insertion of spinal instrumentation (pedicle screws). A peripheral nerve action potential can be induced by either direct mechanical stimulation of a spinal nerve root or stimulation with electrical current to sufficiently depolarize the axon membrane to induce a propagating action potential. The pattern and intensity of electrical activity from peripheral muscles innervated by the nerves "at risk" might indicate impingement of a root by instrumentation placed within the spinal canal. Spinal nerve roots also carry sensory neurons from multiple nerve root levels that allow dermatomal SSEPs to be followed.[29,89] However, the variability, overlap in spatial distribution, and difficulty with acquisition of dermatomal SSEPs render their usefulness limited. As with all spinal cord monitoring modalities, the electrical activity recorded must be interpreted by the technologist, anesthesiologist, and surgeon to determine the significance of the data.

The selection of the muscle group to monitor is determined based on which spinal nerve roots are at risk for irritation or injury. Myogenic activity is measured from a spontaneous or triggered response. With spontaneous myogenic activity, the response is elicited when nerve roots are mechanically irritated. The summed activity recorded from several muscle fibers becomes the CMAP. For a normal patient, no recording evidence of spontaneous activity is generated. Patients with preexisting nerve root irritation or neuromuscular disease often have recordings with low-amplitude periodic firing patterns. Mechanically irritated neurons release short

bursts of activity that can last a fraction of a second or longer trains of activity that can last up to several minutes. The more sustained the train of activity is, the greater is the likelihood of nerve root impingement.

Direct nerve root stimulation is used to determine the stimulation thresholds of nerve roots placed at risk during screw placement.[90–98] The bone of the pedicle has higher impedance to current flow than does direct contact of metal on nerve root or dura. Likewise, blood or other tissue fluid provides less impedance than does the intact pedicle. Therefore, it is possible to determine "stimulation thresholds." If no electromyographic activity is observed at stimulation intensities below these thresholds, it is reasonable to assume that a stimulated pedicle screw is completely contained within an intact pedicle. With the use of constant current stimulation,[14,58,86,99–101] various parameters have been developed to determine a breach in pedicle anatomy. Typically, the intensity of stimulation is gradually increased from 0 mA with a rate from 2.3 to 4.7 Hz and a current duration from 50 to 300 ms until a threshold is reached, at which reliable and reproducible electromyographic responses are elicited (Fig. 8-4). In a prospective study of 90 patients receiving 512 pedicle screws, Glassman et al.[102] showed that a 15-mA stimulation threshold protocol provided 98% confidence of a well-positioned screw and that a threshold between 10 and 15 mA decreased the confidence level to 87%. Electromyographic responses elicited below the predetermined "warning threshold" should prompt consideration for careful screw evaluation to ensure proper placement.

RELIABILITY OF NERVE ROOT STIMULATION

Although theoretically sound in nature, the use of pedicle screw stimulation monitoring is susceptible to several factors, both technical and physiologic, that can confound the results. The degree of pharmacologic muscle relaxation at the time of stimulation can lead to false-negative outcomes. Although not as sensitive as cortical or transcranial MEPs, direct nerve root stimulation evoked potentials can be significantly elevated or unobtainable.[24,65] Current shunting can lead to false-positive and false-negative readings. With a breach in the pedicle wall, blood (a low impedance carrier) fills the opened space and provides an accessory pathway for current to exit. If that breach is near an exiting nerve root, a significantly lower current will trigger a myogenic response (false positive). However, if the breach is not in contact with the exiting nerve root or near the stud of the pedicle screw, less current will exit through the pedicle wall and the amount of current needed to depolarize the nerve root will be falsely elevated. Physiologic factors, including chronic nerve compression, diabetes, and even core body temperature, might further falsely elevate the warning threshold, leading to potentially false-negative outcomes.[103–107]

PRACTICAL APPLICATION

Two facts must be kept in mind regarding the use of electrophysiologic monitoring in the setting of spinal trauma: (1) the awake alert and responsive patient serves as the best source of information relating to spinal cord and nerve root
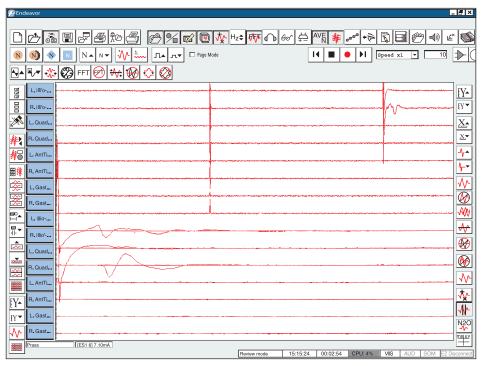

Fig. 8-4 Indirect stimulation of nerve roots via pedicle screw instrumentation. Graded increases in electrical current generate a motor action potential once bony cortical impedance or threshold has been exceeded.

function, and (2) it is difficult to overestimate the potential for neurologic damage to occur in the setting of acute instability related to traumatic injury. Neurologic injury occurs after hospital admission in a significant proportion of patients. Therefore, any manipulation of a patient with spine injury (transport, positioning) is most safely accomplished with an awake, alert, cooperative patient. Whenever possible, anesthesia should be induced on the definitive operating table to minimize the chance of injury during transfer from a stretcher after induction. Using a "turning frame" type of bed allows for prone positioning without unnecessary motion. In patients with extreme spinal instability but without significant neurologic deficit, awake intubation, either nasotracheal or with fiberoptic guidance, can minimize neck motion. Once intubation has been accomplished, the neurologic examination can be repeated in a cooperative patient to ensure that there has been no change with intubation. Definitive positioning in the prone position can even be accomplished with the patient awake.

The choice of which electrophysiologic monitoring to use is determined by the nature of the injury and the portion of the spine involved. Monitoring SSEPs in a patient undergoing surgery for low lumbar fracture is of little usefulness; little risk to spinal cord integrity is present. However, it is probably prudent to obtain baseline recordings for SSEP and MEP monitoring before definitive positioning when these modalities are to be used. Once again, it is important to communicate early with the anesthesia team to be sure that pharmacologic muscle relaxants are limited so that baseline recordings can be obtained soon after anesthetic induction.

As outlined previously, multiple factors can degrade the quality of the signals obtained during the surgical procedure. Aside from the effects of various inhalational anesthetic agents and muscle relaxants, hypotension and body temperature can have negative effects on signal integrity. In addition, hypotension might have deleterious effects on spinal cord injury because automatic regulation of spinal cord perfusion is disrupted after spinal cord injury. Likewise, hypothermia, already a problem in the trauma situation, can exacerbate coagulation abnormalities. Therefore, every effort should be made to keep the patient's temperature and blood pressure as close to normal as possible. If degradation of the evoked potentials occurs, these two factors need to be examined carefully, as does the anesthetic being used. Careful analysis of any surgical maneuver that might have caused the change can be undertaken simultaneously so that the underlying cause can be corrected as quickly as possible. Often, simply allowing the blood pressure to rise or modifying the concentration of inhalational agent can reverse the change. Spinal shock generally involves decompensated loss of peripheral vascular resistance, and, as such, peripheral vasoconstricting agents are appropriate to reverse hypotension, although hypovolemia secondary to blood loss must always be considered and corrected as well.

Should the patient present with new or worsening neurologic deficit after surgery despite failure to identify changes in monitoring signals intraoperatively, advanced imaging (computed tomography, magnetic resonance imaging) should be performed as soon as the patient's physiologic status permits. The decision to return to the operating room to revise or remove instrumentation must be individualized and based on the particular circumstances of each case. However, the point remains that no amount of intraoperative monitoring can substitute for a careful postoperative neurologic examination.

CONCLUSION

Neurophysiologic intraoperative monitoring during spinal surgery is a dynamic process of complex information gathering that aids in real-time clinical decision making. It is vital to maintain an open line of communication among the technologist, anesthesiologist, and surgeon. Few published reports of the usefulness of SSEP and MEP in the trauma patient are available in the literature, and the techniques of intraoperative monitoring have largely been applied empirically. Much knowledge remains to be gathered regarding the efficacy and safety of intraoperative monitoring in the polytrauma setting. Specifically, the limitations from potential confounding variables, unique physiology in a trauma patient, and standardization of warning thresholds must be carefully scrutinized.

As knowledge of neurophysiology and anatomy has been refined during the past decades, we have seen concurrent improvement in the accuracy and efficacy of neuromonitoring. The combined use of SSEPs, MEPs, and electromyography has allowed surgeons to proceed with more awareness of the spinal instrumentation and its relation to surrounding neural structures. The impact of intraoperative monitoring to warn the surgeon of impending neurologic injury has been immeasurable. Much remains to be investigated in neuromonitoring, and the surgeon must always be aware of the limitations of any of the intraoperative methods. However, the usefulness of intraoperative neurophysiologic monitoring is indisputable.

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Mechanisms of Injury in the Cervical Spine: Concepts, Pathomechanics, Classifications, Instability, and Clinical Applications

INTRODUCTION

The cervical spine is the most mobile portion of the vertebral column and serves to protect the highest segment of the spinal cord as it connects with the brainstem. The cervical spine allows for articulated, balanced support of a relatively large cranium on the torso under normal physiologic stresses. However, under abnormal forces, the cervical spine can be compared with a medieval catapult with its projectile being tied down, preventing its release. Bony fractures and ligamentous injuries result in unique patterns that are distinguished by the structure and architecture of the cervical spine. Once considered untreatable ailments, these injuries now can be managed effectively under current classification systems and treatment options.

CONCEPTS

Cervical spine injuries generally can be conceptualized according to region, type, and age.[1–4] The regions of the cervical spine are subdivided into the upper and the lower cervical spine. The types of cervical spine injuries are grouped into bony fractures and ligamentous disruption. The age is divided between adult and pediatric. Further subcategorization occurs for injuries at the occipital condyles, C1/C2 vertebrae, and the C3 to C7 vertebrae. These are described based on radiographic findings or biomechanical considerations.

UPPER CERVICAL SPINE

The upper cervical spine, also referred to as the cranio-cervical or occipito-cervical articulation, involves the skull base, atlas (C1), axis (C2), and numerous stabilizing ligaments.[1] The skull base is made up of the clivus, occipital condyles, and the foramen magnum. The stabilizing ligaments include the transverse ligament, the alar ligaments, the cruciate ligament, and the tectorial membrane. Of the three types of bones that make up the upper cervical spine, each is different in shape and size. The heavy cranium only formally articulates with the spine at two points—the occipital condyles—with the other powerful ligaments providing added stability. The atlas is the smallest bone in the spine, lacking both a vertebral body and intervertebral disks. It serves to support the occiput and is part of the anchor for the highly specialized transverse ligament. In contradistinction, the axis is the largest of the cervical vertebrae, and it is the first vertebrae with posterior facet joints. An extremely high degree of mobility—nearly 50% of rotation and 50% of flexion/extension—is conferred over two articulations. Significant rotation occurs at C1-C2, while substantial flexion/extension occurs at the occiput-C1 articulation.[1,5–7] Unique fractures and ligamentous injuries occur at the occiput, atlas, and axis. These injuries collectively comprise 15% of adult cervical spine trauma. Approximately 9% to 26% of patients with upper cervical spine injuries also harbor lower cervical spine trauma.[5,8,9]

LOWER CERVICAL SPINE

The lower cervical spine, also called the subaxial spine, refers to the C3 through C7 vertebral levels. More redundant in shape and size, the subaxial cervical spine consists of nearly identical vertebral bodies stacked on top of one another with intervening disks and facet joints. In an adult, the C6-C7 interspace acts as the fulcrum of cervical motion.[2] Injuries of the subaxial spine may include both bony fractures and ligamentous injury, and they are often described based on the mechanism of injury and on the vector forces at time of injury. Because there is less

reliance on ligamentous structures in the lower cervical spine compared to the upper cervical spine, injuries of the lower cervical spine tend to include more bony fractures than ligamentous injuries.[8,10]

BONY INJURY

Each bony vertebral segment serves to bear weight via the vertebral body, restrict motion via the facets or the dens, anchor ligaments via the lateral masses or the transverse ligament, and protect the spinal cord via the laminae. Bony fractures can be detected on plain radiographs, but are better delineated on computed tomography. Fractures usually indicate that significant force was present at time of injury. However, osteopenia or osteoporosis can lead to fractures under normal physiologic forces and loads. Fractures may be simple or complex (comminuted) and may be associated with instability. The presence of one fracture should not only prompt the search for another noncontiguous fracture, but also raise the concern for ligamentous injury.[8,9,11] Fractures have traditionally been classified by pattern and biomechanical etiology (Table 9-1).

LIGAMENTOUS INJURY

To maintain stability, the upper cervical spine relies on several ligaments, including the transverse ligament, the alar ligaments, the cruciate ligament, the tectorial membrane, the anterior longitudinal ligament (ALL), the posterior longitudinal

TABLE **9-1**   Fracture Classifications

UPPER CERVICAL TYPES	FEATURES	MECHANISM	EXAMPLES	STABILITY
OCCIPTIOCERVICAL DISLOCATION[1,29]				
Type I	Anterior dislocation of condyle on C1 lateral mass	Anterior translation with slight distraction	Slipping beneath seat belt getting chin caught (in children) Hanging	All unstable* Types I/III require traction reduction followed by immobilization and/or fusion Traction is contra-indicated in Type II injures
Type IIA	Vertical distraction of C0-C1 joint >2 mm	Axial/vertical distraction		
Type IIB	Vertical distraction of C1-C2 joint >2 mm	Axial/vertical distraction		
Type III	Wedge-shaped avulsion fracture of condyle by alar ligament, usually unilateral	Posterior translation with slight distraction		
OCCIPITAL CONDYLEFRACTURE[21]				
Type I	Unilateral impacted fracture, no displacement	Axial load with slight lateral bend	Rollover Heavy object dropped on head	Usually stable, immobilize in hard collar/halo
Type II	Same as type I with basilar or occipital fracture, or bilateral condyle fractures	Severe axial load with lateral bend		
Type III	Wedge-shaped avulsion fracture of condyle by alar ligament	Lateral bend with rotation		Unstable
ATLAS FRACTURES[6,23]				
Posterior arch fracture	Isolated fracture of posterior arch	Hyperextension with axial load	Rollover Heavy object dropped on head	Stable
Ring fracture (AKA Jefferson's fracture)	Fracture of both anterior and posterior arches	Axial load		Stable or unstable†
Lateral mass fracture	Isolated fracture of C1 lateral mass	Axial load with lateral bending		Stable or unstable†
Avulsion fracture	Avulsion fracture of medial lateral mass by transverse ligament	Axial load or C1-C2 translation		Unstable
ODONTOID FRACTURES[24,25]				
Type I	Avulsion fracture of dens tip by alar ligament	Lateral bend with rotation	Rapid deceleration	Stable
Type II	Complete fracture through the odontoid base	Hyperflexion	Blow to back of head	Unstable
Type IIA	Type II with marked comminution	Hyperflexion		Unstable
Type III	Fracture of C2 body	Hyperflexion		Stable

TABLE 9-1   Fracture Classifications—cont'd

UPPER CERVICAL TYPES	FEATURES	MECHANISM	EXAMPLES	STABILITY
C2 PAR INTERARTICULARIS FRACTURE (AKA HANGMAN)[26,27]				
Type I	Fracture at pars with <3 mm translation and no angulation	Hyperextension with compression	Hitting windshield/ dashboard in MVA	Unstable
Type II	Pars fracture with >3 mm translation and significant angulation	Hyperextension with axial load followed by translation and deceleration		Unstable
Type IIA	More angulation, less translation than type II	Flexion distraction	Rapid deceleration	Unstable
Type III	Type I fracture with facet dislocation	Flexion distraction followed by extension	with shoulder belt Whiplash injury	Unstable
ATLANTO-AXIAL SUBLUXATION (DEFINED AS ADI >3 mm FOR ADULTS AND >5 mm FOR CHILDREN)[12,13,15]				
Ligamentous	Ruptured transverse ligament	Flexion with anterior translation or axial load	Restrained MVA with rapid deceleration (shoulder belt with airbag)	Unstable
Rotational (types I-IV)	Unilateral subluxation of C1 on C2	Rotation with ligamentous laxity due to inflammation	Antecedent URI/ infection	Unstable

*Power's ratio: Distance from basion to posterior arch of C1 divided by distance from anterior arch of C1 to opisthion is normally less than 1. Values greater than 1 indicate instability.

†Spence's rule: If the total overhang of the C1 lateral masses on C2 on an open-mouth x-ray or computed tomography scan is greater than 7 mm, the transverse ligament is likely injured and is unstable. If the total overhang is less than 7 mm, the transverse ligament is likely intact, and stability depends on the preservation of anatomic alignment.

ADI, Atlanto-dental interval (distance from dorsal aspect of anterior arch of C1 to ventral aspect of dens); MVA, motor vehicle accident; URI, upper respiratory infection.

ligament (PLL), and the joint capsules. Failure of these ligaments, even in the absence of a fracture, may lead to instability.[12–15] Ligaments can fail as a result of avulsion off its bony attachment, excessive stretching leading to increased laxity, or direct rupture. Infants and young children are particularly prone to injury because of the laxity of their ligaments, which can be stretched excessively.[16,17]

The ligamentous structures of the lower cervical spine, which contribute to spinal stability, include the ALL, PLL, intervertebral disks, facet joint capsules, interspinous ligament, and ligamentum flavum.[2,10,18]

PHYSEAL INJURIES

A special class of pediatric injury is fracture through or involving the epiphyseal growth plate. Salter Harris type I fractures are those that traverse the epiphyseal plate horizontally in the plane of the growth plate and are considered unstable in the spine. They occur in young children and often are difficult to diagnose because the bones recoil to normal alignment after injury.[16] Salter Harris types II and III fractures are those that cross the epiphyseal plate at an approximately perpendicular angle and occur in adolescents when the plate begins to close. Types II and III fractures are defined in long bones by involvement of the metaphysis or epiphysis and cannot be distinguished in the spine. They are generally stable fractures in the spine.

ADULT INJURY

The most common causes of spinal injuries in adults are motor vehicle accidents, falls, and athletic injuries (about 30% each).[10] Younger, more active adults sustain higher energy injuries (e.g., high-speed accidents and sports injuries), whereas older adults sustain significant injury with lower-energy impacts (e.g., low-speed collision or fall from low height). In older patients, preexisting stenosis or spondylosis also contributes to higher rates of neurologic deficits with cervical spine trauma.[19,20]

Specific types of fractures are commonly recognized because of their frequency in the population (Table 9-1). For instance, occipital condyle fractures result from lateral

bending and axial loading and they may easily be overlooked in the presence of more obvious injuries.[21,22] Jefferson fractures are ring fractures of C1 in which both anterior and posterior arches are disrupted because of excessive axial load.[23] Dens fractures are those that violate the odontoid process.[24,25] Hangman fractures involve breakage of the C2 pars interarticularis and represent a detachment of the anterior and posterior elements at C2.[26,27] Teardrop fractures are small anterior avulsion injuries of a vertebral body. Facet injuries are primarily ligamentous injuries, but may include varying degrees of bony fractures. Facet injuries may occur unilaterally or bilaterally and can manifest as "perched" or "locked" facets. These injuries are generally considered unstable and require open or closed reduction with internal fixation. A clay shoveler's fracture is an avulsion fracture of a spinous process and generally is considered stable.

PEDIATRIC INJURY

The most common causes of spinal injuries in children vary depending on age.[16,28] Among neonates and infants, birth trauma (from breech or forceps-assisted delivery) and abuse are most often cited.[16,28] Play or sports injury and car accidents are the dominant causes in children younger than 15 years.[16]

Pediatric cervical spine injuries are predominantly ligamentous in nature and thus more injuries occur at sites where soft tissue support provides major contributions to stability. Laxity of the transverse ligament allows for more translation at C1-C2 and often can stretch without breaking but still lead to neurologic injury.[15] In addition, joint articulations are more flat and smooth at young ages and allow for increased motion. This is especially true at the occipital condyle-C1 lateral mass joints.[16] This concept applies to virtually all ligaments and joint capsules in the pediatric spine, and it helps explain why 15% of cervical spine injuries in children are occipital-cervical dislocations (compared to only 6% in adults).[4] The relatively heavy mass of the head and lack of neck muscle control in neonates and infants especially predispose them to injury if shaken or abused.

Cervical spine injuries in infants and children can be divided into three categories: infantile, young juvenile, and old juvenile. Infantile injury is defined by injury prior to the development of adequate head control.[16] Mechanisms include traction or torsion injury during birth and violent shaking during abuse.[17] These injuries are often occult on radiographs because bony fractures are rare. Injury is often a result of overstretched ligaments that return to their original position.

Young juvenile injury refers to injury after the development of adequate head control but younger than 8 years.[16] These injuries are primarily above C4, with C2-C3 being the most commonly affected level.[16] In children younger than 8 years, the fulcrum of flexion/extension is centered in the upper cervical spine mainly at the C2-C3 disk space. The posterior facet joints are still oriented horizontally and the unco-verterbral joints (joints of Luschka) are largely underdeveloped at this age. Thus, little translational restraint at C2-C3 is offered during extreme flexion and extension, making subluxation easier.

Old juvenile injury includes those in children older than 8 years.[16] The fulcrum of cervical motion is midcervical during this age, and most primary ossification centers (except the tip of the dens) have completed fusion by 8 to 10 years. The posterior facets begin to take adult shape by age 10 and the ligaments of upper cervical spine begin to stiffen significantly. These injuries take on more of an adult appearance, and sports-related injuries become prevalent.

PATHOMECHANICS

The cervical spine bears the least amount of weight in the spine and confers the highest degree of mobility. Normal physiologic motion can be described by segmental articulations beginning with the occiput and ending with the first thoracic vertebrae. Seven types of force comprise the pathologic vectors leading to fracture or injury: compression, distraction, flexion, extension, translation, lateral bending, and rotation. These forces are often present in combination and yield unique injury patterns based on spinal localization.

OCCIPITAL-CERVICAL (C0-C1)

The occipital-cervical articulation is responsible for 13 to 21 degrees of flexion and extension, and this constitutes approximately 50% of the flexion-extension arc normally achieved by the cervical spine.[6,18] The occiput-C1 junction is limited in rotation and lateral bending, with only 7 degrees of rotation and 5 degrees of lateral bending conferred.[6]

Because the attachment of the occiput to the atlas relies mainly on ligamentous structures, and no occipital-atlanto disk exists, the types of injuries seen at the occipital-C1 junction usually are compression and distraction injuries.[6,29,30] Various measurement methods, such as the Power's ratio and Chamberlain's line, have been used to determine thresholds for which atlanto-occipital dislocation occurs, and their use depends on physician preference. Atlanto-occipital dislocation tends to occur more often in children than in adults because natural ligamentous laxity at young ages combined with the presence of a relatively large head and high fulcrum in the cervical spine. A compressive force, however, can produce a burst of the ring of C1, or Jefferson's fracture.

ATLANTO-AXIAL (C1-C2)

The atlanto-axial articulation is responsible for 45 degrees of rotation—nearly 50% of the cervical rotation arc—and only 10 degrees of flexion extension.[7,14,15,26] There is virtually no lateral bending motion at the C1-C2 articulation. The main translational stabilizer of the atlanto-axial articulation is the

transverse ligament, which is twice as strong as all the other stabilizing ligaments.[14,15,24] Fusion of the C1-C2 vertebral levels predicts a significant loss of absolute rotational motion, except in younger patients, who generally are not limited by degenerative changes and age-associated ligament stiffening.[7,31] Because of compensatory motion in the cervical spine, total rotation is lost in only 13% in patients younger than 20 years, 25% in patients between ages 20 to 40, and 28% in patients older than 40 after C1-C2 fusion.[12,13]

The atlanto-axial joint also relies heavily on ligamentous attachments for translational and distractive stability, and thus it is prone to injury resulting in C1-C2 subluxation from fracture of the dens or rupture of the transverse ligament. The atlanto-dental interval (ADI) is the most common radiographic measurement used to assess subluxation and is abnormal in adults at greater than 3 mm and in children at greater than 5 mm. Because the C2 pars interarticularis also serves to transfer translational control from the odontoid-transverse ligament complex to the posterior aspect of C3, fractures can commonly occur at the pars interarticularis. This is known as a hangman fracture.[14,24,26,32–34] The mechanism of this tends to be compression with flexion, commonly from motor vehicle accidents.

SUBAXIAL CERVICAL SPINE (C3-C7)

The remainder of the cervical spine consists of nearly identical intervertebral segments starting with the C2-C3 intervertebral segment, ending at C7-T1. Approximately 8 to 17 degrees of flexion-extension, less than 10 degrees of rotation, and 4 to 11 degrees of lateral bending is afforded per level in the subaxial cervical spine.[2,30] The vertebral bodies provide axial load support, while the "shingled" posterior facets provide translational stability. The intervertebral disks and facet joints limit excessive compression-distraction, while allowing for flexion-extension and some rotation.

The fulcrum of motion in a normal adult cervical spine is at the C5-C6 and C6-C7 disk space and explains why these are the most commonly injured and herniated cervical disks.[4,30,35] The subaxial spine is prone to injury from virtually all pathologic vectors of force.

Because the cervical spinal canal is large, canal compromise can be tolerated to a certain degree. This benefit is especially true in the upper cervical spine, where nearly half of the spinal canal consists of cerebrospinal fluid (CSF). However, the vital function of the upper cervical spinal cord for respiration makes cord injuries extremely severe and potentially lethal (such as in atlanto-occipital dislocation). In the lower cervical spine, the spinal cord occupies more spinal canal volume as it approaches the thoracic portion, and on average, CSF space only constitutes less than one-third of the spinal canal volume. Commonly, significant traumatic brain injury is associated with both upper and lower cervical spine injuries and often complicates management of these severely injured patients.[1,2,9,10]

CLASSIFICATIONS OF INJURIES

Because similar upper cervical spinal injuries can result from several different pathomechanical insults, these injuries are categorized individually based on (1) level or location of injury and (2) bony or ligamentous involvement. Table 9-1 summarized these distinct entities and their common classifications, injury mechanisms, and degree of stability.

Lower cervical spinal injuries are more patterned, based on pathomechanical vectors of force and the position of the neck during injury. These vectors of force include flexion, extension, compression, distraction, lateral bending, and rotation. These forces are commonly present in combination, resulting in characteristic fractures and injuries. Flexion with compression leads to anterior "wedge-shaped" fractures, whereas flexion with distraction results in facet dislocation. Unilateral facet dislocation occurs with rotation and flexion. Extension injuries commonly result in facet fracture.

INSTABILITY

Spinal instability is classically defined as the inability to maintain normal anatomic relationships resulting in (1) damage to neural elements, (2) incapacitating pain, or (3) deformity under physiologic loads and forces.[1,2,4] An alternative popular definition emphasizes functional anatomy: Any disruption of the anterior load-bearing column, posterior tension band, vertebral body subluxation or dislocation, or spinal canal compromise is considered unstable.[4] In general, greater than 50% vertebral body height loss, 3.5 mm translation, or 11 degrees angulation per level is considered abnormal and unstable.[3,4,10,30] For the lower cervical spine, a point system has been described incorporating relevant factors to score spinal instability[4,36] (Table 9-2).

The three-column model of the spine, well-described by Denis for thoracolumbar trauma, has also been applied to the

TABLE **9-2**   Diagnosis of Clinical Instability in the Lower Cervical Spine: A Point System

ELEMENT	POINT VALUE
Anterior elements destroyed or unable to function	2
Posterior elements destroyed or unable to function	2
Relative sagittal plane translation >3.5 mm	2
Relative sagittal plane rotation >11 degrees	2
Positive stretch test	2
Nerve root damage	1
Abnormal disk narrowing	1
Dangerous loading anticipated	1

Scores of 5 or greater indicate clinical instablility.[4]

cervical spine.[35,37–39] In this model, the spine is divided into three columns: anterior, middle, and posterior. The anterior column consists of the anterior longitudinal ligament and the anterior half of the vertebral body. The middle column is made up of the posterior half of the vertebral body and posterior longitudinal ligament, and the posterior column consists of the pedicle or pars interarticularis, posterior facets and facet joints, interspinous ligament, and ligamentum flavum. Disruption of a single column can be considered stable. Disruption of any two columns including the middle column is considered unstable.[34,39] Involvement of all three columns is automatically considered unstable. Although this model can be translated for use in classifying lower cervical spine fractures, upper cervical spine fractures do not conform well and are usually considered individually with specialized criteria for instability.

CLINICAL APPLICATIONS

HISTORY, EXAMINATION, AND LABORATORY TESTS

The evaluation of a patient with a cervical spine injury begins with a comprehensive history, with special attention to the mechanism of injury and related factors. For motor vehicle and pedestrian accidents, information regarding the estimated speed of the vehicle, use of a seatbelt by the patient, and condition of the vehicle (i.e., cracked windshield, rollover) can help to predict certain spinal injuries. For fall injuries, the height from which the fall occurred should be determined. For diving and sports-related accidents, information regarding head, neck, and body position at time of impact can be useful. A complete neurologic examination, including mental status and cranial nerve examination in patients with suspected head injury, should be performed. The motor examination should test all major muscle groups bilaterally. The sensory examination should include testing for light touch and pinprick or temperature sensation for the entire body to assess for a sensory level. Chronic injury is evidenced by increased reflex jerks and clonus, whereas decreased or absent reflex jerks point to either hyperacute injury or peripheral nerve damage. Rectal tone may still be present even with significant injury and must be ascertained in each patient. Central cord syndrome is the most common incomplete cervical spinal cord injury and is hallmarked by disproportionately greater motor impairment in upper compared with lower extremities. The bulbocavernosus reflex should be evaluated and its presence helps determine an incomplete spinal cord injury. Close monitoring of the patient's vital signs is also critical for patients with suspected spinal injury. Significant injury to the cervical and thoracic spinal cord and column can lead to neurogenic spinal shock because of loss of sympathetic tone on the circulation. A low blood pressure without reflexive tachycardia in the presence of neurologic deficit suggests spinal shock.

During the initial workup, standard laboratory tests for surgery are requisite and include complete blood count, chemistry panel, coagulation profile, and type and cross match for blood transfusion.

RADIOGRAPHIC IMAGING

Radiographic evaluation for cervical spine injury begins with x-ray plain films. Lateral and anterior-posterior (AP) images can be quickly obtained in the emergency room but must clearly visualize all cervical levels including the bottom of C7 to be adequate. Swimmer's view plain films can be obtained to help visualize obscured lower cervical anatomy. Open-mouth view AP images can be used to assess C1 ring fractures and odontoid fractures.

If an abnormality is found on plain films, or if x-ray imaging is inadequate, computed tomography (CT) is now widely available to assess the extent and configuration of bony injury. CT scans with two-dimensional (2D) sagittal or coronal reconstruction of 1 or 3 mm slices are extremely useful for operative planning and should be obtained in cases requiring surgery. However, intervertebral disk and ligament injury is inadequately visualized with CT and requires further imaging with magnetic resonance imaging (MRI).

MRI imaging should be obtained in patients with neurologic deficits and normal x-ray or CT imaging to rule out an underlying epidural hematoma or disk herniation causing significant neurologic symptoms. Although not requisite for all spinal injuries requiring surgery, cases with severe deformity requiring significant reduction should have preoperative MRI imaging.

In cases with normal radiographic findings and an intact neurologic examination but significant pain on presentation, flexion/extension x-ray after neck muscle spasms have resolved is recommended to evaluate instability from ligamentous injury. Flexion/extension films should not be performed in cases with clearly identified severe injuries or neurologic deficit for fear of exacerbating spinal cord injury in an unstable spine.

INTERVENTIONS

Unstable injuries require immediate immobilization. External orthoses include hard collars, halo vests, and Minerva vests and can be used as a bridge to surgery. Depending on the type of fracture, external orthoses may be all that is needed. The goals of surgery include spinal canal decompression with reduction of any deformity followed by rigid internal fixation. Traction can be used prior to surgery to help reduce severe injuries but should not be used if severe distraction injury is suspected. Traction should be performed with the patient awake to allow for monitoring of neurologic changes and lateral x-rays should be obtained prior to the addition of more weight. Once in the operating room, prevention of hypotension during intubation or

induction of anesthesia is critical and prophylactic antibiotics should be used routinely. It is important to remember that induction of anesthesia produces relaxation of the cervical musculature, and extreme care must be taken when performing traction. Aggressive physical rehabilitation and early mobilization both are extremely important interventions postoperatively.

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Nonoperative Treatment of Cervical Fractures: Cervical Orthoses and Cranioskeletal Traction in Patients with Cervical Spine Fractures

INTRODUCTION

Cervical orthoses are appropriate for three major applications. First, they limit abnormal vertebral displacement associated with spinal fractures. Second, they minimize painful spinal motion associated with degenerative disorders. Third, they supplement or substitute for internal fixation after reconstruction of the cervical spine.

In the setting of cervical spine fractures, the immediate objective is to reduce the risk of additional injury to the spinal cord. This goal is achieved by providing mechanical support to the neck and by limiting the neck range of motion either with cranioskeletal traction or cervical orthoses. Failure to adequately stabilize the injured cervical spine can result in neurologic deterioration with possible devastating consequences. In addition, chronic pain syndromes might ensue.

Cervical orthoses can be divided into two categories: collars and post appliances. Alternatively, they can be categorized by the craniocaudal extent of the orthosis: cervico-thoracic, occipitocervical, and occipito-cervico-thoracic. All cervical orthoses remind the wearer to restrict head and neck motion. Moreover, a well-designed orthosis should impose forces to position the head and to limit flexion, extension, rotation, and/or lateral motion of the head and cervical

spine. Ideally, the orthosis should be designed to reduce the load on the cervical spine by supporting a portion of the weight of the head.

Craniocervical traction was first introduced in 1933 by Crutchfield for the management of facet dislocations causing subluxations and subsequent spinal cord impingement. The original traction device has been modified by several persons. Gardner introduced the Gardner-Wells tongs in 1973, and that device is most widely used in clinical practice for short-term traction for cervical spine injuries. Spine surgeons must have some familiarity with the role and limitations of cervical orthoses and cranioskeletal traction.

IN-THE-FIELD IMMOBILIZATION

Cervical spine immobilization is one of the most often performed prehospital procedures after traumatic accidents in which a known injury mechanism can lead to cervical fractures or ligament injuries. Extrication collars (e.g., Stifneck) must be carried in a reasonable quantity and range of sizes in a relatively small amount of space in emergency medical services vehicles. Therefore, they usually are folded flat in shrink-wrapped packages. The extrication collars can easily lead to pressure ulceration with prolonged use, especially in an obtunded patient. If cervical clearance is not possible within 24 to 48 hours, the extrication collar should be replaced with a more appropriate orthosis, such as an Aspen collar, Miami J, or PMT collar.

For initial immobilization, the old-fashioned sandbag and tape combination might work equally well. Podolsky et al. systematically examined the efficacy of cervical spine immobilization methods and found that tape and sandbag immobilization is more effective than any single orthotic device. In addition, this method is effective in achieving cervical immobilization when used alone or in combination with a semirigid orthosis.[1,2]

CERVICAL BIOMECHANICS

When used for spinal instability, the most important criteria for cervical orthosis are maximum restraint of motion and static posture. Therefore, understanding the biomechanics of

the cervical spine is crucial. The cervical spine produces a remarkable broad range of motion: 145 degrees of flexion and extension, 180 degrees of axial rotation, and 90 degrees of lateral flexion.[3] The degree of flexion, extension, lateral bending and axial rotation at the individual cervical segments varies (Fig. 10-1). The absolute values used in the clinical evaluation for cervical range of motion are as follows: flexion (70 degrees ± 10 degrees), extension (75 degrees ± 10 degrees), lateral bending (45 degrees ± 10 degrees), and axial rotation (75 degrees ± 10 degrees).[2−5]

CHOOSING AN ORTHOTIC DEVICE

A wide spectrum of cervical orthoses is available to accommodate the different pathologic abnormalities related to the cervical spine. At one end of the spectrum is the soft collar, providing minimal mechanical restraint, and at the other end is the halo, which provides marked limitation in all directions. No orthosis completely limits motion in any plane (Table 10-1).

An important consideration in choosing any cervical orthosis is understanding its ability to restrict motion at each craniocaudal level in the cervical spine. At a minimum, cervical orthoses provide proprioceptive feedback to the alert patient, leading to more favorable cervical position and active restriction of motion. Total contact orthoses increase pressure inside the interior cavities of the body. The increased intracavitary pressures transfers load from the functional spine units to the surrounding soft tissues and therefore reduces the load borne by the involved spinal segments.[6,7]

Recent data have been published by various groups who used biomechanical analysis to determine the effectiveness of common cervical collars and cervicothoracic orthoses.[5,8,9] Although these studies are limited in that normal partici-

pants or cadavers were included, they provide useful information for selecting a specific orthoses. The degrees of flexion and extension in SOMI, four-poster cervical, and cervicothoracic braces compared with a normal unbraced cervical spine have been evaluated (Figs. 10-2, *A* and *B*). A well-documented phenomenon called *snaking* occurs at the upper cervical spine. At the level of the occiput and C1, more flexion is permitted when any brace is applied.

Lunsford et al.[5] compared four cervical collars (Philadelphia, Miami J, Malibu, and Newport extended wear) and determined that the Malibu collar was superior in restricting motion in flexion, extension, and axial rotation. Although not as effective as the poster-type or halo-vest orthoses, collar orthoses were shown to be effective in limiting cervical motion by 40% to 60%.

Semirigid cervical collars and cervical thoracic orthoses often are used as an adjunct to surgical stabilization. In contrast, semirigid cervical orthoses are insufficient for immobilization of unstable fractures. The halo is the only cervical orthosis indicated for unstable cervical fractures. The halo vest can be applied after reduction and traction if the clinical scenario does not allow definitive surgical stabilization and for patients who might not be acceptable surgical candidates.[10−13]

In 1981, the effectiveness of halo vests on patients with injured spines was evaluated. The data showed that flexion and extension could be limited to 7.5 degrees (11.7% of normal), total lateral bending to 4.1 degrees (8.4% of normal), and rotation to 2.2 degrees (2.4% of normal).[14] These results clearly distinguish the halo as the most effective means for restricting cervical motion in all three planes of motion. Figure 10-3 depicts the effectiveness of all classes of cervical orthoses, including the halo. The halo is effective at reducing the "snake effect" at the upper segments.[15]

Table 4.3.   Middle and lower cervical spine range-of-motion limits and representative values (C2-T1)

	COMBINED FLEXION/EXTENSION (+Θx)		LATERAL BENDING (ONE SIDE) (Θz)		AXIAL ROTATION (ONE SIDE) (Θy)	
	LIMITS OF RANGES (DEG)	REPRESENTATIVE ANGLE (DEG)	LIMITS OF RANGES (DEG)	REPRESENTATIVE ANGLE (DEG)	LIMITS OF RANGES (DEG)	REPRESENTATIVE ANGLE (DEG)
Interspace						
Middle						
C2-3	5–16	10	11–20	10	0–10	3
C3-4	7–26	15	9–15	11	3–10	7
C4-5	13–29	20	0–16	11	1–12	7
Lower						
C5-6	13–29	20	0–16	8	2–12	7
C6-7	6–26	17	0–17	7	2–10	6
C7-T1	4–7	9	0–17	4	0–7	2

From White AA, Panjabi MM: Clinical biomechanics of the spine, ed 2, Philadelphia, 1990, JB Lippincott.

Fig. 10-1 Relative range of motion of the cervical spine at specific levels below C1. (From Lusardi M, Nielsen C, eds: Spinal Orthoses. Boston, Butterworth-Heinemann, 2000.)

TABLE 10-1   Commonly Used Orthotics for Treatment of Cervical Spine Injuries

CERVICAL LEVEL DIAGNOSIS	ORTHOSIS
C0	
Occipitocervical dislocation and subluxation	ORIF, halo
C1	
Posterior arch	Collar
Jefferson fractures	
<7 mm displacement	Collar, CTO
>7 mm displacement	Halo
Ruptured mid-transverse ligament	Surgery
C2	
Type I	Collar
Type II	Halo or surgery
Type III	Halo
Atlantoaxial Rotatory Deformities	
Reducible	Collar, CTO
Unreducible	Traction or surgery
Hangman's Fracture	
Type I	Collar
Type II	Collar/halo
Type III	Surgery
C3-C7	
Flexion/Compression Fractures	
Stable	Collar/halo
Unstable	Surgery
Burst Fractures	
Neurologically intact/stable fracture pattern	Halo or CTO
Neurologic deficit/unstable fracture pattern	Surgery
Facet Dislocations	
Unilateral	Tongs/halo, then surgery
Bilateral	Tongs/halo, then surgery
Distraction-Extension Injuries (Intact Ligament/Disk)	
Without spinal cord compression	Halo/surgery
With spinal cord compression	Surgery
Ruptured ligaments/disk/fracture	Surgery
Compression-Extension Injuries	
Nondisplaced	Collar/CTO
Displaced	Surgery

CTO, cervicotnoracic orthosis.
Adapted from Rothman and Simeone: The Cervical Spine, 4th ed. 1982, Table 7.2.

CERVICAL ORTHOSES

SOFT COLLARS

A soft foam collar provides minimal control of motion in the cervical spine (Fig. 10-4). Flexion, extension, and lateral bending is limited by 5% to 15%. Rotation is limited by 10% to 17%. It provides feedback through the sensory system that consciously or subconsciously causes the patient to adopt a more corrective or appropriate position of the neck. The soft collar increases a patient's awareness of an injured area through contact with the skin and reminds the patient to self-restrict motion of the involved area. It keeps the neck warm, and patients find it extremely comfortable.[7]

A soft collar can be used when the following benefits are desired for the patient: warmth, psychological comfort, support to the head during acute neck pain, relief with minor muscle spasm associated with spondylosis, and relief in cervical strain. A soft collar, however, is contraindicated for bone or ligament injury to the neck. It is made of polyurethane foam with a cotton stockinet covering and a Velcro closure. It is prefabricated, and minimal adjustments are required. The orthosis is sized based on the circumference of the neck. The height varies from 2.5 to 4.5 inches, and the standard height is 3 inches. It is available in adult and pediatric sizes. The average cost is $50.[6]

HARD COLLARS

Hard cervical collars are indicated for stable midcervical spine injuries, strain, sprain, and stable bone or ligament injury. In a multitrauma setting, they often are used for unconscious patients until the neck can be cleared. Caution must be used when an orthosis is placed on a comatose patient with elevated intracranial pressure because even a correctly fitted collar can further raise intracranial pressure.[16] Solely relying on hard collars to treat unstable cervical spine fractures might lead to pseudoarthrosis or arthrodesis with loss of physiologic lordosis. Even worse, the patient is at risk for spinal cord injury during that period. Use of hard collars after fusion surgery or after a more rigid orthosis, such as a halo vest, is removed might be helpful. Most hard collars allow thoracic extension to increase motion restriction between C6 and T2.

PHILADELPHIA COLLAR

The Philadelphia collar is an extended wear collar (Fig. 10-5). It provides more coverage of the head and neck than does the soft collar and is an example of a cervical orthosis. It is a two-piece, prefabricated collar made out of polyethylene foam with rigid Kydex, a high-temperature-molded plastic. The anterior and posterior piece of the collar extends to the mandible and limits flexion. The posterior portion extends over the occiput, providing some limitation of extension. The inferior portion of
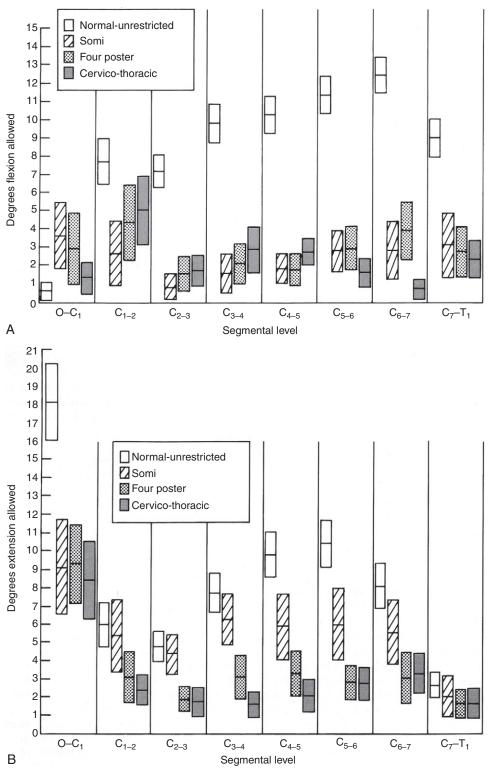
Fig. 10-2 *A,* Degrees of flexion allowed in the unbraced cervical spine, SOMI, four-poster cervical, and cervicotho-racic braces. *B,* Degrees of extension allowed in these same orthoses. Note the "snaking" that occurs at the upper cervical spine. The study was conducted with normal subjects. (From Rothman R, Simeone F, eds: The Spine, 2nd ed. Philadelphia, Saunders, 1982.)
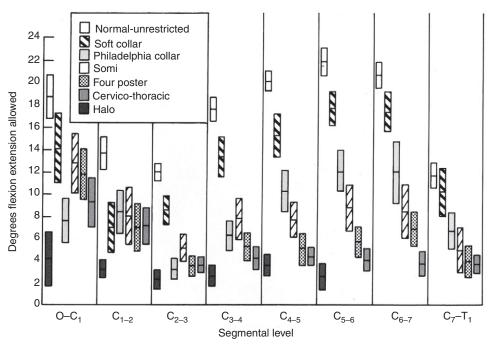
Fig. 10-3 Effectiveness of all classes of cervical orthoses, and the halo. Note that this study included some patients who had surgical arthrodesis.

Fig. 10-4 Soft cervical collar made with polyurethane foam and covered with a cotton stockinette and can be adjusted using the Velcro closure.

the collar extends to the upper portion of the thorax, again providing resistance to flexion and extension.[5,6]

The Philadelphia collar is similar to the soft collar in that it provides proprioceptive feedback to limit neck motion and retains body heat. The reinforcement provided by the anterior and posterior plastic strips enables this orthosis to provide greater restriction to cervical flexion, extension, rotation, and lateral bending than do the soft collars. In addition, the anterior and posterior plastic reinforcing strips permit more selective adjustments of head position than those provided by soft collars. This collar limits lateral flexion to approximately 45 degrees, forward flexion to 40 degrees, extension to 34 degrees, and axial rotation to 32 degrees.[5] It is available in various adult and pediatric sizes. It is comfortable for most patients to wear and therefore is associated with increased patient compliance. It costs approximately $125.[6]

MIAMI J COLLAR

The Miami J collar is a semirigid cervical orthosis that is similar in composition to the Philadelphia collar. It is a two-piece system made of polyethylene and a soft washable lining (Fig. 10-6). It has an anterior opening for a tracheostomy. Velcro straps make for easy application and adjustments. The Miami J collar also can be heated and molded for a more custom fit. A thoracic extension can be added to increase support and treat lower cervical (C6−T2) fractures. It limits flexion and extension by 55% to 75%, rotation by 70%, and lateral bending by 60%.[5,17] It is available in various sizes and costs approximately $150.[6]

ASPEN COLLAR

The Aspen collar is similar to the Miami J and the Malibu collars in that it is a two-piece system made of polyethylene with a soft foam liner and it has an anterior opening for a

A

B

Fig. 10-5 *A,* Oblique view of adult Philadelphia collar. *B,* Lateral view with pediatric patient. The Philadelphia collar has a posterior portion that extends over the occiput and the inferior portion of the collar extends to the upper portion of the thorax. This collar comes in a variety of sizes.

Fig. 10-6 Miami J collar has removable, washable inserts and an anterior extension for greater limitation of cervical flexion. It is the most commonly used collar for pediatric patients for long-term wear.

tracheostomy. It is a semi-rigid head-cervical orthosis with Velcro straps for easy application and adjustments (Fig. 10-7). This collar limits flexion and extension by approximately 55% to 60%, rotation by 60%, and lateral bending by 60%. This collar comes in a variety of adult and pediatric sizes. It costs approximately $160.[3,5,6,8]

PMT CERVMAX COLLAR

The Cervmax collar is one of the newer cervical orthoses on the market and therefore has not been subjected to any rigorous testing outside of that conducted by its manufacturer. It has a unique V-back design to provide maximum occipital support (Fig. 10-8). It has a smaller shell in the chin area, designed to reduce skin irritation. It supports the mandible to control coronal flexion and extension. It is equipped with the patented Coolmax liner, which wicks moisture away from the skin. This collar also comes in different colors for pediatric sizing and aesthetic appeal. It has undergone motion restriction testing by the PMT Corporation, and the results suggest that it performs similarly to the Aspen and Miami J collars. It costs approximately $58.

CERVICOTHORACIC ORTHOSES

Cervicothoracic orthoses provide greater motion restriction in the middle to lower cervical spine as compared with cervical collars. This is achieved by the added pressure on the body. Cervicothoracic orthoses restrict cervical flexion, extension, lateral bending, and rotation. They do not, however,

Fig. 10-7 Aspen collar (*A*), in oblique, lateral (*B*), and posterior (*C*) views. It has removable, washable inserts. It can be converted to a two-poster or four-poster CTO.

reduce axial loading. In general, this type of orthosis provides less motion restriction of the upper cervical spine.[3]

Hartman's group[18] used cinefluoroscopic data to compare the soft collar, semirigid collar, four-poster cervical orthosis, and Guilford two-poster cervical orthosis. They evaluated the orthoses for motion restriction in all three planes of motion and found the Guilford design to be superior.

Johnson et al.[19] used roentgenography to evaluate the effectiveness of five cervical orthoses in restricting sagittal motion in normal participants. They concluded that the cervicothoracic orthosis with rigid connections between the anterior and posterior components and fixation to the chest is superior.

Gavin et al.[8] combined three-dimensional head motion by using an optoelectronic motion measurement system, electromyography, and video fluoroscopy to test the limitations provided by the Miami J and Aspen collars. Twenty normal participants were included in the study, which found that the Miami J and Aspen collars were similar in their capability to provide intersegmental motion limitation in flexion and extension. The Aspen collar, however, seemed to outperform the Miami J at C5-C6.

ASPEN CERVICOTHORACIC ORTHOSES

Aspen cervicothoracic orthoses come in two types (Fig. 10-9): with rigid thoracic extensions anteriorly (two-post Aspen) and with thoracic extensions anteriorly as well as posteriorly (four-post Aspen). The four-post Aspen cervicothoracic orthosis was found to restrict flexion by 99% and extension by 77%. Gavin et al.[8] found that the four-post cervicothoracic orthosis seemed to outperform all collar orthoses and the two-post cervicothoracic orthosis in extension at all cervical levels. Cervicothoracic orthoses currently are indicated for minimally unstable fractures. Cervicothoracic orthosis is recommended for stabilizing

Fig. 10-8 PMT Cervmax collar has opening for tracheostomy and a Coolmax liner, which wicks away moisture from the skin.

Fig. 10-9 Aspen CTO system is a combination of the Aspen collar attached to a vest with either two or four posts attached.

below C4. Gavin et al. found that Aspen cervicothoracic orthoses significantly restrict head motion. No statistically significant difference was found between the two- and four-post setups.

MINERVA

The Minerva brace, a custom cervicothoracic device, encloses the entire posterior skull with a band for the head and extends downward to the inferior costal margin (Fig. 10-10). If greater control of cervical and thoracic motion is desired, the brace may be extended to a pelvic girdle. It allows for stabilizing forces under the chin and around the occiput to restrict flexion and extension, lateral motion, and rotation of the head and cervical spine. It partially supports the weight of the head, thus reducing the axial load on the cervical spine. Depending on the caudal application of extensions, this device also limits thoracic motion to varying degrees. The sizes range from adult to infant, and can be customized.

STERNOOCCIPITOMANDIBULAR ORTHOSIS

The sternooccipitomandibular orthosis (SOMI) consists of three components: the sternal yoke, the occipital component, and the mandibular component (Fig. 10-11). This orthosis is

a post-type device that differs from other designs because the uprights that maintain the position of the occipital support arise anteriorly from a sternal plate. This arrangement allows the SOMI to be applied to the supine patient and permits the wearer to lie on the back comfortably. Additionally, the single anterior upright with its attached mandibular support can be quickly and easily removed from the sternal plate. This affords the patient opportunities to eat, wash, or shave while remaining in a supine, semirecumbent position. Thus, it is ideal for patients who are bedridden. This is the orthosis of choice when dealing with pediatric patients with injury to C4 and above. The SOMI also can provide a modification consisting of a prefabricated polyethylene and Dacron skull strap, which can be substituted for the mandibular support. The strap snaps onto the occipital support and encircles the upper skull. It allows the patient to chew and is used primarily to permit eating and not as the definitive treatment. There are also fixtures available to enable the SOMI to be attached to a custom made spinal jacket, thus increasing overall orthotic control of the vertebral column.[3]

The SOMI is relatively comfortable to wear. As with most orthoses, proper fitting and adjustment are necessary for

Fig. 10-10 The Minerva CTO encloses the occiput and supports the chin to effectively reduce the weight of the head on the cervical spine. It has an adjustable vest for added support of the cervicothoracic junction.
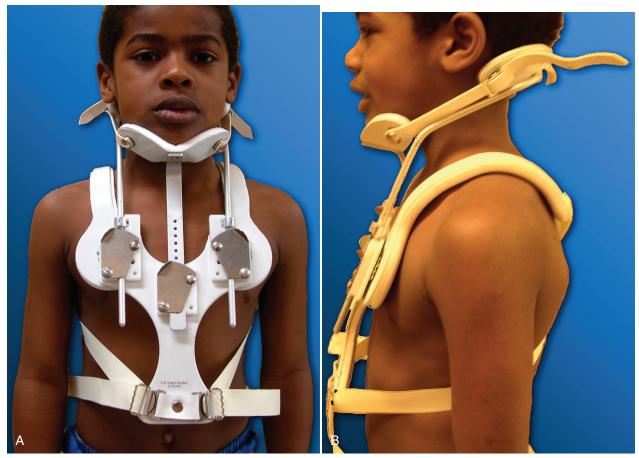
Fig. 10-11 Sternooccipitomandibular orthosis (SOMI), shown in anterior *(A)* and lateral *(B)* views, consists of three components: the sternal yoke, the occipital component, and the mandibular component.

motion restriction. An incorrectly applied SOMI brace may provide minimal restriction. It is very effective in controlling flexion at the atlantoaxial and C2-C3 segments. The SOMI is indicated for immobilization in atlantoaxial instability secondary to rheumatoid arthritis and immobilization for neural arch fractures of C2 because flexion causes instability. Flexion and extension are restricted by 70% to 75% per level from C1-2 to C7-T1. Lateral bending is limited by 35% and rotation is limited by 60% to 65%.[7,8] Average cost for a SOMI brace is $480.[6]

EFFECTIVE USE OF ORTHOSES

Three important factors should be remembered in order for a semirigid collar or Cervico thoracic orthosis (CTO) to be maximally effective. First, a snug fit must be maintained. If the collar or device is loosened or altered in any way after the original fitting, the ability of the device to limit neck motion diminishes significantly. This would be similar to not wearing the collar at all.

Second, there are complications related to wearing collars and CTOs. Because of the firm fit and total contact with the skin of the mandible, occiput, and superior thorax, patients are at risk for skin irritation and breakdown. Therefore, it is of utmost importance for the patient and caregivers to be educated regarding proper collar hygiene. The collar liner should be removed and washed every 24 hours. It is necessary for the prescribing physician or treating nurse to order a replacement liner. This allows the patient to wear a clean liner while the other is being washed and air-dried. Also, it may be necessary to provide patients who are bedridden some time out of the collar to minimize pressure on the occiput. The occiput seems to be the most vulnerable area for pressure sores or wound breakdown in patients who may have had posterior stabilization. There are other complications associated with the application and wearing of cervical orthoses: local pain, muscle atrophy, decrease in vital capacity, psychological dependence, and loss of fracture reduction or displacement.[17,20–22]

Third, neutral alignment should be maintained even when the patient is supine or sleeping. It is recommended that a single pillow be used versus multiple pillows behind the neck to prevent unnecessary flexion of the neck. This could produce discomfort and possibly increase the risk of skin breakdown.

DISCONTINUING ORTHOSES

When a semirigid cervical collar is going to be discontinued, it is preferable to wean the patient. This is probably a better practice than abruptly discontinuing the collar as it gives the patient time to strengthen weak neck muscles and regain normal proprioception. This process may also lessen psychological fear associated with the abrupt loss of the brace as a means of protection. The practitioner may devise a schedule, allowing the patient to wear the collar less and less over a 2-week period until it is completely discontinued. Alternatively, the practioner can suggest a soft collar as a substitute to the semirigid orthosis.

THE HALO

The halo fixator is composed of graphite composite materials. It is magnetic resonance imaging (MRI) compatible because it uses a limiting graphite composite ring and titanium hardware. It requires a single torque driver for all adjustments. The vest has adjustable belt-style buckles and straps. There are also interlocking tabs that reduce relative movement between the front and back halves of the vest. There is a cardiac crease in the front half of the vest, which allows for rapid access to the chest area. There are vest styles that come with an inflatable air bladder for adjusting anterior and posterior compression for a more custom fit. The halo and vest come in adult and pediatric sizes. The current estimated cost of a halo ring and vest outfit is $1800.

SIZING

The patient has to be sized for an appropriate fitting halo and vest system. The following four measurements are required: (1) head circumference, (2) xiphoid circumference, (3) waist circumference, and (4) shoulder to iliac crest. The halo ring comes in open back or closed back, and ranges in size from xx-small to xx-large. Selecting the halo ring is done by measuring the circumference of the head 1 cm above the eyebrow and the ear. One should choose the larger size if measurements fall in between sizes.

Choosing the correct vest size is done by measuring the circumference of the chest at the xiphoid process. If the patient is between two sizes, then measure the circumference of the waist and the vertical distance for the top of the shoulder to the iliac crest. The vest sizes include small to xx-large.

SUPPLIES AND PREPARATION

The physician and orthotist must also have additional supplies available for applying the halo. These items include 10-mL syringe, 22-Ga × 1½-inch needle, local anesthetic, sterile gloves, Betadine prep, 4 × 4 pads, scissors, razor, and a flashlight. Placing the halo and vest is a three-person job and requires a team approach. One of the practioners should be knowledgeable of the nature of the cervical instability and ideally stand at the head of the bed positioned for holding the head during placement of the halo.

Choose the proper size halo ring and vest using measurements and sizing chart for the product. The patient must be supine with the head supported. The open back halo and traction rings are designed for application directly on the bed. Head pins should be sterile because this decreases the risk of introducing bacteria into the skin. The pin sites must be chosen to avoid potential complications, including damage to the temporalis muscle and zygomaticotemporal nerve.

Placement should also allow the patient to wear glasses. Optimal placement of anterior halo pins is in the anterolateral aspect of the skull, 1 cm superior to the eyebrow or orbital rim, above the lateral two thirds of the orbit, and below the equator of the skull (Figs. 10-12, *A* and *B*).

This area is considered the safe zone as placement of the pin in this area prevents displacement into the orbit and migration of the pin upward. Lateral to the safe zone is the temporalis muscle and fossa and the zygomaticotemporal nerve. If the pin were to migrate into the temporalis muscle, this could cause pain with mastication or speaking. Also the temporal bone is thinnest here and could easily be penetrated or cause loosening of the pin. The zygomaticotemporal nerve is a small cutaneous nerve in this region and if damaged, could lead to numbness, pain, or paresthesia. The medial structures to be avoided include the supraorbital and supratrochlear nerves and the frontal sinus. Placing pins in the lateral third of the orbit will avoid injury to these structures and lessen the risk of penetrating the frontal sinus.

Placement of posterior pins is less risky because there are no important neuromuscular structures. The skull is also more uniform posteriorly and thicker. Positions are easily chosen if the skull is divided into a clock at the equator with the anterior midline position at 12 o'clock and the posterior midline position at 6 o'clock. Correct position of the posterior pins is at the 4 o'clock and the 8 o'clock positions. Optimally, pin sites should be inferior to the equator of the skull, but approximately, 1 cm superior to the tip of the ear. Figure 10-13 illustrates the correct position of all four pins in the adult.

HALO PLACEMENT

The patient should be prepared by explaining the procedure and obtaining informed consent. The patient should also receive light sedation with 5 to 10 mg of diazepam (Valium) and 1 to 2 mg of morphine unless there are allergies to these medications. If there are allergies, then an alternate muscle relaxant or light sedative can be chosen. The patient should be awake enough for ongoing neurologic assessments.

The halo ring has several positioning pins, that help to place the halo in optimal position prior to pin insertion. These pins have plastic positioning pads that contact the skin
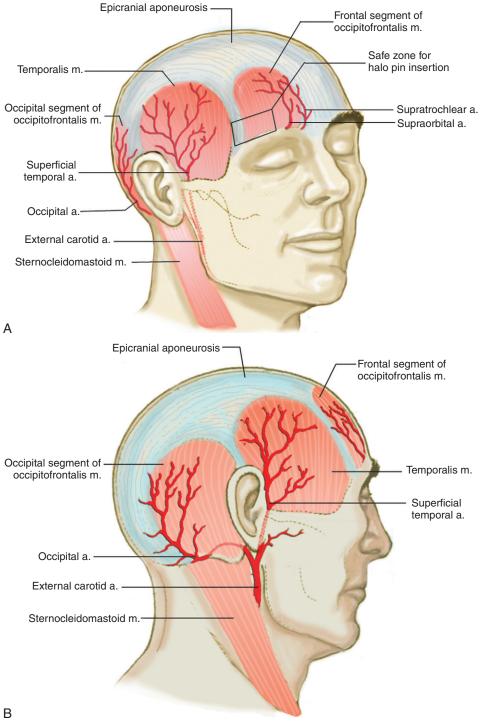
Fig. 10-12 *A,* "Safe zone" for placement of anterior pins, which avoids damage to supratrochlear and supraorbital nerves. *B,* Posterolateral view of the head demonstrating relevant anatomy around the area of pin placement.

for comfort. The pin sites should be shaved in 1- to 2-inch diameter using the razor. The area should be prepped with Betadine or other skin prep solution.

The patient is directed to keep his or her eyes closed during placement of the anterior pins. This prevents skin and eyebrow tenting, which can hinder the patient from closing the eyelids and can cause necrosis of the skin. One should inject local anesthetic to the skin and more importantly down to the periosteum. The needle can be placed directly through the halo ring, or, alternatively, an assistant can shine a light directed through the chosen pinhole to illuminate the correct location on the skin.
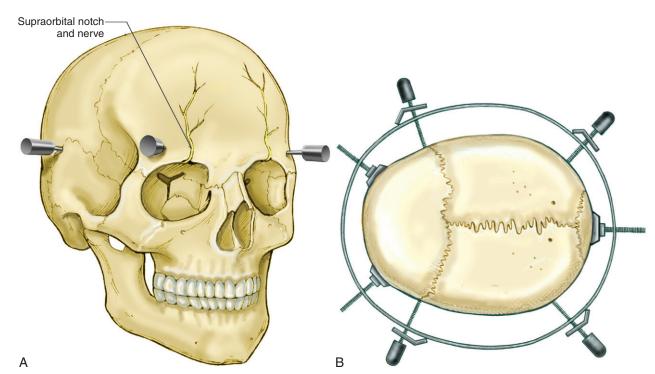
Fig. 10-13 *A,* Anterior and poster pin placement. *B,* Halo and pin placement with positioning pins in place.

Keeping the halo pins sterile, they are inserted into the appropriate holes. The pins can be finger tightened at this time until they reach the outer table of the skull. Using the torque driver, the pins can be further tightened in 2-pound increments up to 6 pounds in the adult. One should tighten pins that are directly diagonal to each other in an alternate fashion. Next one should place the pin lock nut and tighten until it is flush with the ring. The brass positioning pins and pads can be removed. The pins should be checked after 24 to 48 hours and tightened to 8 pounds.

Next, one should apply the head block to either side of the halo ring using Allen screws and washers. The bulk of the head block should be above the equator of the skull. The head block should be adjusted so that the long axis is perpendicular to bed.

THE VEST AND RODS

The posterior shell of the vest is applied by log rolling the patient. Careful attention should be used in keeping the neck aligned. One should slide the posterior shell underneath the patient as much as possible. Then the patient is rolled to the opposite side to adjust the vest. The lower portion of the posterior vest should be in line with the lower portion of the rib cage. Now, the anterior section of the vest can be applied. The lower portion of the anterior vest is positioned at the waistline. One should secure the vest with straps and buckles making sure that the fit is adequate.

The posterior rods are applied first. One should not tighten posterior rods until the anterior rods have been placed. Figure 10-14 shows the completed halo, vest, and rod assembly. Once the setup is complete, adjustments can be made to achieve the desired amount of flexion or extension of the neck. A follow up x-ray should be taken after placement of halo and vest. The patient should also be monitored closely for complications, including dysphagia.

INDICATIONS FOR HALO PLACEMENT

The halo fixator is commonly used for instability of the upper cervical spine, especially C1-C2, supplemental stabilization after surgical fixation, and for unstable fractures of the mid- and lower-cervical spine. Relative contraindications include unstable skull fractures, abraded skin at potential pin sites, osteoporosis, myelopathy, and poor patient compliance.[21]

COMPLICATIONS OF HALO PLACEMENT

The halo device is an extremely efficacious treatment for certain cervical spine pathology; however, its use is associated with minor and major complications including pin loosening, pin site infections, loss of reduction, ring migration, severe pin discomfort, swallowing difficulties, dural puncture, pin site bleeding, nerve injury, scarring, skin breakdown, and patient intolerance. Improper pin placement can cause damage to the supratrochlear, supraorbital, and zygomaticotemporal nerves.[3,21] By understanding the principles of halo placement and adher-

Fig. 10-14 This is the PMT halo and vest device. There are several brands of halo devices, but the configurations are all similar.

ence to strict application principles, these complications can be kept to a minimum.

PIN SITE CARE

The incidence of halo pin infection is approximately 20%. Pin site infection is best prevented by simple cleaning with half percent hydrogen peroxide twice daily.[23] If drainage and erythema persist at any pin site, bacterial cultures should be obtained and the patient should be treated with appropriate antibiotics. In most cases this takes care of the problem, however some infections will progress to form an abscess. In these cases, the pin should be removed and the pin placed in an adjacent site.

CRANIOSKELETAL TRACTION

Cranioskeletal traction is used to reduce spinal deformity from trauma or other etiology such as rheumatoid arthritis. Once reduction is achieved, weight on the traction is lowered

to maintain reduction and alignment until definitive surgical treatment. By urgently reducing traumatic spinal deformity, the possibility of neurologic recovery is enhanced and secondary injury to the spinal cord is minimized.[24]

Cranioskeletal traction can be achieved with Gardner-Wells tongs or with the halo ring. Gardner-Wells tongs consist of a C-shaped metal ring and sharp threaded spring-loaded pins as shown in Figure 10-15. A smaller S-shaped ring accompanies the ring for attachment of a rope and weights.[3] The patient should be on a gurney or a hospital bed that allows for the attachment of bars, pulley, and weights. A cervical collar should be maintained until the traction is applied. Local anesthesia is given prior to insertion of the pins. The pins are inserted 1 cm above the pinna of the ear in line with the external auditory canal. The pins are tightened until the indicator protrudes 1 mm. The pins may require 24-hour evaluation for retightening. The S-shaped hook is attached to the ring and a rope knotted and extended over the bed and connected to the weights.

If the patient is awake, alert, and cooperative to give reliable feedback during cranioskeletal traction, further neurologic deficit may be avoided. There are no hard and fast rules regarding the upper limit of weighted traction. The current
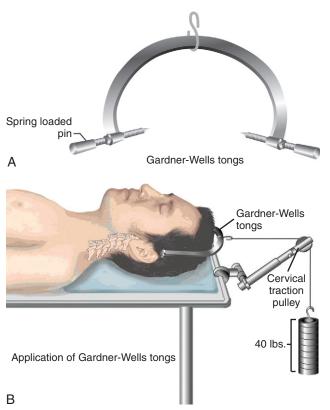
Fig. 10-15 A, Gardner-Wells tongs, a C-shaped ring with spring loaded pins that are placed approximately 1 cm above the pinna of the ear. B, Gardner-Wells tongs in place with weighted traction in an awake and alert patient.

practice is to add 10 pounds and up to 10 pounds per vertebral level may be used. This rule does not apply for atlantooccipital dislocation where even 5 pounds of weight may cause overdistraction. Weight can be increased by 5 to 10 pounds every 10 to 15 minutes until reduction is achieved. The patient's neurologic status should be evaluated after each incremental load. If there is a change in the neurologic examination, the weight must be reduced until the baseline neurologic examination is obtained.[3,25] A static x-ray or fluoroscopy should be used to follow each added weight. A fluoroscopy unit increases efficiency during the process because one is able to evaluate changes in alignment immediately. This helps avoid overdistraction. This procedure should be performed in the emergency room (ER) or preferably an intensive care unit (ICU) where vital signs can be monitored continuously.

COMPLICATIONS OF CRANIOSKELETAL TRACTION

Complications of application of tongs include pin failure, intervertebral disk herniation,[26] vertebral artery dissection,[27] overdistraction, iatrogenic fractures,[28] and acute quadriplegia.[29]

PEDIATRIC CERVICAL SPINE

Cervical spine injuries in children are rare, approximately 1% of all pediatric blunt injuries and 10% of all cervical spine injuries.[30] This may be attributed to increased elasticity and flexibility of the immature spine, with the upper cervical spine being most vulnerable in children younger than 8 years and the lower cervical spine being the most vulnerable to injury in children 8 years and older. Major anatomic and biomechanical differences exist between the adult and pediatric cervical spine until age 8 years.[31] Large head to body proportions, underdeveloped neck musculature, and facet anatomy increase the risk of cervical spine injury in children. By age 12, the spine has mostly matured. Understanding these differences can aid in the management of pediatric spine injuries in the acute period.

When immobilizing a child on a firm backboard, even with a semirigid collar, it is recommended that the torso be elevated to prevent kyphotic angulation and deformity. Alternatively a recess for the occiput can be used.[32] Most spine injuries in children can be managed nonoperatively. Halo fixation or skeletal traction, followed by brace immobilization results in a 95% rate of fracture healing when applicable.[33] From our review, all cervical orthoses have similar biomechanics in adults and children.

CONCLUSION

Choosing the correct orthoses in cervical trauma requires a multidisciplinary approach. First, the injury must be clearly defined from the mechanism of injury, examination, and imaging studies. This should be followed by asking "what is the best treatment for this injury for this patient." If an orthosis is a desirable treatment modality, attention must be given to the design, fitting, and follow-up of the orthoses. The best orthosis is well fitted and delivers the desired mechanism of action to achieve an optimal treatment result with the most comfort possible and at the lowest cost.

A well-trained orthotist, certified by the American Board for Certification in Orthotics and Prosthetics, is a valuable resource for physicians. Orthotists can recommend the proper orthosis, custom fit the desired orthoses, and assist with cranioskeletal traction. To minimize noncompliance, patient education, including the patient's family is extremely important. A line of communication between the patient and a health care professional should be maintained regarding the use of the orthosis even after discharge from the hospital.

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Cervical Whiplash Injuries

INTRODUCTION

Whiplash has traditionally been defined as an acute cervical soft tissue injury, consisting of hyperextension and/or hyperflexion forces, resulting in musculoligamentous sprain or strain.[1,2] The diagnosis typically excludes fractures or dislocations of the cervical spine, head injury, or alterations in consciousness. Plain radiographs and magnetic resonance imaging (MRI) scans of the cervical spine are typically negative.[3–8] The neck pain and stiffness that can result is often part of a constellation of symptoms now termed whiplash-associated disorder (WAD), which can also include arm pain and paresthesias, headache, dizziness, visual disturbances, problems with concentration and memory, and psychological illness. Temporomandibular dysfunction is included by many, but whether or not a rear-end impact can cause supraphysiologic forces has been questioned in recent biomechanical studies.[9–11]

In 1995, The Quebec Task Force (QTF) defined whiplash as a general acceleration-deceleration mechanism of energy transfer to the neck and developed a classification for WAD that is commonly used today. A grade 1 injury results in neck pain, stiffness, or tenderness, without physical signs; grade 2 injuries represent neck complaints with musculoskeletal signs such as decreased range of motion (ROM) and point tenderness; grade 3 consists of additional complaints such as a sense of heaviness, arm muscle fatigue, and paresthesias; grade 4 adds fractures and dislocations, which are very rare from a whiplash mechanism without head impact[12] and are often excluded by others when discussing WAD (Table 11-1). According to the QTF, whiplash is an acute, self-limited injury.

The incidence of WAD varies considerably by culture and country, probably because of numerous issues such as injury expectations, the possibility of secondary gain, and the ability to capture and follow patients. WAD is more common in those with pretraumatic neck pain, low education level, and

female gender, with WAD grades 2 to 3 being the most common.[13] The incidence of WAD is more than 100 times higher in American women workers (14.5 per 1000),[14] than in the general population of New Zealand (0.1 per 1000).[15] Interestingly, demolition derby drivers do not seem to suffer from WAD despite an average career total of 1900 collisions per driver, many of which are at high speeds.[16]

Several other studies bring the overall diagnosis and understanding of whiplash into question. A study of 36 consecutive patients involved in high-energy motor vehicle accidents (MVAs) with ISS greater than 16 and no diagnosed cervical spine injury showed similar results. Only two patients had any neck symptoms, headaches, or paresthesias, and both had resolutions of their symptoms by 2 months.[17] Furthermore, no association has been found between velocity or force of injury and incidence of WAD, and yet, 65% of all rear-end impacts have minimal vehicle damage, but significant neck symptoms seem to occur[18] despite lower forces than in amusement park bumper cars.[19]

Many authors have discussed the gender differences in WAD, although the cause is not clear. Females are more than twice as likely to suffer a whiplash injury as males and are more likely to suffer from chronic symptoms.[20,21] Biomechanical testing suggests that female spines may have higher shear loads at the lower cervical facet joints, during the anterior distraction and posterior compression motions seen in a whiplash injury, than male spines.[22] This could result in increased activation of mechanoreceptors and nociceptive nerve fibers.[23] Female sex hormones have a negative impact on collagen content of the joint capsule,[24] and there is a difference in the layout of cartilage in the facet joints of males and females.[25] A study looking at the individual motion segments in women after WAD found significantly increased rotational moments in the C3-C4 and C4-C5 segments, as well as increased translational motion at C3-C4 and C5-C6, prompting the authors to suggest specific exercises to target the deep segmental muscles to try to correct the abnormal motion.[26] Women also generally have a smaller width to their spinal canal than men. This may be important given that the canal pinch diameters (CPDs) were found to be significantly narrower during the hyperextension phase of whiplash at

TABLE **11-1**   Quebec Task Force Classification of Whiplash-Associated Disorder

WHIPLASH ASSOCIATED DISORDER CLASSIFICATIONS	SIGNS AND SYMPTOMS
Grade 1	Neck pain, tenderness, and stiffness
Grade 2	Above + decreased ROM and point tenderness
Grade 3	Above + arm heaviness, fatigue, and paresthesias
Grade 4	Above + cervical fractures and/or dislocations

ROM, Range of motion.

accelerations at 3.5 g and above. The narrowest CPD was at C5-C6, where it was 3.5 mm narrower compared with the neutral posture diameter.[27] This could result in a central cord syndrome or other cord injury in patients with narrow spinal canals. Acute and chronic whiplash injury patients with higher levels of pain and disability have sensory hypersensitivity to a variety of stimuli, suggestive of central nervous system sensitization.[28,29] This has been substantiated further by Pettersson et al.[30] who found that patients with chronic symptoms after whiplash had a significantly smaller width to their spinal canal than the patients who improved by 12 months.

Neck ROM is significantly reduced, with the exception of extension, in whiplash patients compared with controls. This is especially true in those that suffered the injury within a year compared with those with longer duration. Lateral bending and left rotation are particularly affected. No correlation has been found between ROM and WAD score, type of collision, or pain side.[31,32] However, reduced ROM 3 months after whiplash injury is a predictor of persistent pain and disability.[33] Whether the decreased ROM is due to muscle spasm or muscle dysfunction in an attempt to splint a subtly injured spine is unknown. Ninety percent of whiplash sufferers have been found to have some degree of muscle spasm,[34] and electromyography (EMG) has shown that whiplash patients had a decreased ability to relax the trapezius muscles.[35] Recording of pressure-induced pain in the splenius and trapezius has shown that most patients recover in 4 to 6 weeks' time, with a minority showing no improvement at 6 weeks.[36] Interestingly, MRI studies of patients 2 days to 3 weeks after injury have failed to show evidence of muscle damage.[4–6]

Symptoms lasting longer than 6 months are considered chronic and fall outside the diagnosis of acute WAD. Some then use the diagnosis of chronic whiplash or chronic myofascial pain. However, whether or not chronic whiplash injury even exists has been a source of much debate. Kwan and Friel[37] published a review and methodologic critique of the literature with the intent of supporting "chronic whiplash injury." They identified 37 articles that met their criteria for review and found all contained significant methodologic errors relative to the author's statements regarding chronic whiplash. However, the failure to identify the cause or true incidence rate does not rule out the existence of chronic whiplash or WAD. For patients with neck pain and apparent soft tissue injuries following high-energy trauma, a thorough understanding of the biomechanics of this type of injury will aid in the treatment decisions.

BIOMECHANICS

Biomechanical studies on cadaveric spines and testing on normal volunteers have lead to an increased understanding of the mechanics and injury patterns seen in whiplash injuries, as well as in factors that may reduce their severity. In rear-end collisions there is a forward acceleration of the trunk caused by the impact. The head is effectively left behind, forcing the lower cervical spine into extension as the skull ends up posterior to T1. In cadaveric specimens without protective muscle contractions, the peak intervertebral rotations of C6-C7 and C7-T1 exceed the maximum physiologic extension at a 2.5 g forward acceleration. The upper cervical spine initially remains in flexion, resulting in an S-shaped curve during the time of maximum lower-level extension.[38,39]

The opening stress on the anterior disk of the lower vertebrae and compressive force on the facets is further accentuated by the upward acceleration of T1 that is dependent on seat back angle and worse with a seat back angle of 40 degrees. The sternocleidomastoid muscle contracts eccentrically in an attempt to stabilize the head, followed by activation of the paraspinal muscles. The head and entire cervical spine eventually extend before being accelerated forward, bringing the entire cervical spine into flexion.

Frontal collisions when wearing a three-point seatbelt cause a flexion-distraction force on the cervical spine. This can cause a range of injuries from mild cervical sprains in low-energy impacts, to compression fractures, odontoid fractures, facet fractures and dislocations, and other high-energy trauma in high-speed accidents. The most common significant injuries appear to be either upper cervical injuries such as odontoid fractures and hangman's fractures or lower cervical fractures and dislocations around C6.[40–45]

Prepared occupants in MVAs appear to have lower injury rates than those that are unprepared.[46,47] Experiments designed to address this issue have found that surprised test subjects have higher paraspinal muscle amplitudes and larger angular head accelerations than those that are expecting the impact.[48–51] Similarly, pre-tensing the cervical muscles results in a reduced peak magnitude of response to an acceleration.[52] The change in velocity required to cause symptoms is under investigation but appears to be around 8 km/hour.[53–56] Higher accelerations result in decreased onset time and

peak EMG time for the muscles on the opposite side of impact.[51,57–59]

Finite-element modeling has shown that the major force on the upper and lower cervical spine is shear, and it is directly proportional to impact acceleration. Head restraints do not affect the magnitude of the shear force, but they do decrease intervertebral translation because the head moves more in phase with the torso, which results in decreased injury.[60,61] The pain generators after a rear-end whiplash injury are still under investigation. Whether the specific injury in WAD is in the facet joint or capsule, paraspinous muscles, or disk is still open to debate.

FACET INJURY

One source of pain appears to be the facet joints, which are injured during the extension phase. Autopsy specimens of people would had experienced soft tissue neck injuries have revealed hemarthroses in the facet joints, as well as damage to articular cartilage, capsular tears, and synovial fold displacement.[62–65] Similar damage has been elicited in cadaveric specimens subjected to low-speed rear-impact simulations.[66] However, bone scan studies have not confirmed facet injury.[67,68] More detailed biomechanical studies have shown that peak facet joint compression and sliding are greatest at C4-C5 and exceed the physiologic limits at 3.5 g and 5g, respectively; that capsular ligament strains are greatest at C6-C7 and exceed physiologic strains at 6.5 g; and, although peak facet joint compression occurs at maximum extension, peak ligament strain occurs when the facet joint opens as it begins returning to its neutral position.[69,70] This may result in facet joint injury, capsular injury, or nerve stimulation.

Studies in goats have shown that nerve fibers in facet joint capsules respond to mechanical stimuli, with afterdischarges occurring when the capsule is stretched beyond the physiologic range. Mechanoreceptors and nociceptors have also been identified in human facet capsules.[71] Distending facet capsular ligaments in healthy volunteers with facet joint injections has produced whiplashlike pain patterns.[72] Lord et al.[73] reported that in patients with WAD and headaches, C2-C3 facet blocks relieved the symptoms in 50% of them. The prevalence of lower cervical facet joint pain after whiplash was found to be 49% using placebo-controlled facet joint blocks. Facet block and radiofrequency ablation of facet joint efferent nerves have been shown to be a beneficial treatment in certain patients with WAD by other investigators as well.[73–76]

The effect of head position on facet joint injury has also been studied. Fifty-seven percent of whiplash patients with pain lasting more than 2 years reported that their heads were rotated at the time of impact.[20,47] Furthermore, in the study by Sturzenegger et al.,[47] head position at impact was the only significant correlation with symptom duration. Biomechanical testing on the effect of rotation, or pretorque, on facet capsule strain showed that rotation increases strain magnitudes, could

cause noncatastrophic capsule injury, and may provide a mechanical basis for increased injury or nociceptive stimulation.[71] It should be noted, however, that facet capsular ligaments were not ruptured and that gross failure of the ligaments from low-speed rear-end collisions is unlikely. Siegmund et al.[77] found that capsular strain increases with shear load, but does not vary with axial compressive loading, and that capsular ligaments may be injured during whiplashlike loads of combined shear, bending, and compression.

MUSCLE STRAIN

Some data support the notion that muscle strain is more important than facet injury in WAD. Several studies have shown that the neck does not move beyond physiologic range for low-velocity impacts[78–80] because of eccentric muscle contraction. The contraction begins partly from proprioceptive input before any significant head movement occurs.[81] With low-velocity rear-end impacts, head movement begins 60 milliseconds after impact and ends within 400 milliseconds.[82,83] Muscle contraction begins 60 milliseconds after head movements begins, with the levator scapulae, sternocleidomastoid, and trapezius muscles responding first, before peak head acceleration is reached.[39,78,80,82–86]

EMG studies have shown that in rear impacts the greatest muscle response comes from the sternocleidomastoids, in which they can generate up to 179% of the maximal voluntary contraction (MVC) with accelerations as high as 13.7 m/sec^2,[49] whereas in frontal collisions the trapezii are the most active.[87] The effects of head position and direction of impact have been extensively studied by Kumar et al.[59] If the head is rotated at the time of a rear impact, then the contralateral sternocleidomastoid generates the strongest contraction while the other is protected. In a frontal impact, if the head is turned to the left, the left trapezius generated 77% of its MVC, which was more than double the response of the other muscles. The other muscles that turn the head to the left were active as well, with the right sternocleidomastoid generating 25% of its MVC, and the left splenius 32%. The time to onset and peak EMG for all muscles decreased as the level of acceleration increased.[88]

In lateral impacts with the head and body in neutral position, the greatest muscle response comes from a near maximal contraction of the contralateral splenius capitis muscle.[51,58] If the head is rotated at the time of the lateral impact, then the contralateral sternocleidomastoid experiences a stronger contraction than the ipsilateral one, but the contralateral splenius capitis still experiences the strongest contraction.[89] With right anterolateral accelerations of 13 m/sec^2, and the head rotated to the right, the right trapezius generates 61% of its MVC, and the left sternocleidomastoid 26%. With the head rotated to the left, the right sternocleidomastoid generated 22% of its maximum contraction despite the same direction of impact.[90] The effect of a left lateral impact on the cervical spine when the victim is out of the recommended driving position has

also been studied. With the trunk flexed 45 degrees and laterally flexed 45 degrees, all muscles generated less than 37% of their MVC, even at an acceleration of 13.7 m/sec^2. The right sternocleidomastoid was less active with truck flexion, regardless of lateral bending, than in the upright position,[91] suggesting that flexed posture may lessen the risk of injury to some cervical muscles. Similarly, the cervical muscle response was also found to be reduced when the trunk was simply flexed left or right of neutral during a left anterolateral impact.[92]

CERVICAL DISK

The cervical disks may also be a source of pain in whiplash. Biomechanical testing has also shown supra-physiologic loads on the cervical disks during rear-end type of accelerations. Significant increases over sagittal physiologic levels were first noted in the posterior 150-degree fibers (running posterosuperiorly) of the annulus of C4-C5 during the 3.5 g simulations and reached a maximum of 51.4% at C5-C6 during the 8 g simulation. Peak disk shear strain exceeds sagittal physiologic levels during the 3.5 g simulation and reaches a maximum of 1.0 radian at C5-C6 during the 8 g simulation. Finally, axial deformation exceeds physiologic levels at 3.5 g in the anterior disk region of C2-C3 and C4-C5 and at 6.5 g at C5-C6 in the posterior region.[93] In cadaveric studies without protective muscle contractions, there is increased ROM and neutral zone motion at C5-C6 after a 4.5 g or greater rear-end acceleration, indicating that subcatastrophic ligamentous injury can occur, which could account for chronic symptoms in some patients.[94,95] Clinically, whiplash patients often have positive diskograms that may further implicate the intervertebral disk as the source of pain in some individuals.[96]

Some clinical MRI studies have suggested that whiplash injuries can result in immediate posterior and posterolateral disk herniations, especially at C5-C6.[5,97,98] Others have suggested that whiplash patients are vulnerable to accelerated disk degeneration 5 to 10 years after the accident,[99,100] presumably initiated from annular tears.[101–104] Stimulation of the nerve endings in the outer annulus of the disk may be an etiology of neck pain[105–107] and spasm,[108] and disk degeneration may lead to increased facet arthritis and facet joint pain.[109–111] However, according to Ferrari,[112] MRI studies have failed to find acute disk lesions in whiplash patients with chronic pain.

WHIPLASH IN CHILDREN

Although there is little biomechanical or clinical data on children and whiplash injuries, young children appear to have a greater preponderance of upper cervical injuries in MVAs compared with adults, as well as a higher rate of spinal cord injury without radiographic abnormality.[113–118] This has been attributed to a larger head-to-body ratio, which causes the fulcrum of flexion to be at C2-C3 rather than C5-C6; poorly developed paraspinal musculature; shallow facets and

immature uncinate processes; wedged vertebral bodies; growth centers that are vulnerable to shear forces during rapid deceleration or hyperflexion-extension; and ligamentous laxity.[119–121]

In one retrospective review, 84% of younger children had an injury at C1-C2 versus 31% of older children, and 69% of younger children had ligamentous subluxation compared with 33% of older children.[122] Seventy-two percent of children were unrestrained, however, so they may not have had a true whiplash mechanism.

In a study by Boyd et al.,[123] the rate of WAD in children ages 4 to 16 was found to be 47%. Sixty percent of the patients were symptomatic on presentation. Forty percent became symptomatic the following day. Fifty-five percent were female. Older children trended toward a higher incidence of WAD, but it was not significant in this study. All radiographs of Grade 1 and 2 WAD were read as normal. The mean duration of symptoms was 6.4 days for Grade 1 and 19.7 days for Grade 2. No children developed chronic WAD (pain for more than 2 months). The QTF recommends that radiographs be obtained in all children with Grade 2 symptoms, although it is not known if it is in fact beneficial.[1]

There are a few case reports in the literature detailing cervical flexion injuries in children in MVAs. A common theme in these reports is the lack of proper restraint in the majority of cases; fractures and dislocations of the upper cervical spine, often treated with Roto-rest beds, halos, or fusion; and cases of spinal cord injury without radiographic abnormality (SCIWORA).[124–127]

There are clearly differences between children and adults in the incidence and quality of whiplash injuries. Although it appears adult spines experience more stress during a rear-end whiplash injury in the lower cervical segments, children appear to suffer more injuries in the upper cervical region. The fact that children younger than 8 years are more likely to suffer cranial vertebral junction and upper cervical injuries, odontoid fractures, subluxation without fracture, and SCIWORA have been born out by other retrospective reviews as well.[128–133] Whether these data apply to high-grade rear-end whiplash injuries in properly restrained children is still not known. Caution should certainly be used in young children with loss of consciousness, neck pain, spasm, or torticollis or with signs of trauma to the head or face or with abrasions from seatbelts following low-energy motor vehicle accidents.

TREATMENT

There is much debate over the conservative treatments for whiplash. A review of the literature was performed by the Cochrane Collaboration and published in 2004.[134] Out of five studies comparing passive interventions such as ultrasound, rest, immobilization, and electrical stimulation to placebo, only one met the criteria for a high-quality study.[135] The Cochrane group concluded that there is limited evidence

that would suggest that passive treatment is more effective than no treatment in the short term but not in the long term. Nine studies evaluated the effectiveness of active (meaning no collar and resuming preaccident activities with or without additional exercises) versus passive intervention. Only two were considered high quality. There was no clinically significant improvement in pain in long-term follow-up, but there was faster return-to-work in the active group, according to those high-quality studies. According to the Cochrane Collaboration, most low-quality studies, however, showed positive results in favor of the active interventions and conclude that "rest makes rusty." The latter result has been substantiated by a randomized controlled clinical trial by Rosenfeld et al.[136] who showed that patients who started an active intervention program within 96 hours of injury were more likely to have normal ROM at 3 years than those started after 14 days or who were treated with a soft collar. Similar results were seen by Schnabel et al.[137] in a randomized study with a 6-week follow-up. A retrospective study on home cervical traction by Swezey et al.[138] found that 81% of patients with Grade 1 to Grade 3 WAD had symptomatic relief, although they do not mention how much relief they had or how long it lasted.

Twenty-four hours of high-dose methylprednisolone treatment, started within 8 hours of whiplash injury to all patients presenting after an accident, was found to significantly reduce disabling symptoms, total number of sick days, and sick-leave profile in a small prospective, randomized, double-blind study.[139] However, given the risks of high-dose steroids and the fact that physicians cannot predict which patients will develop WAD, we are hesitant to recommend this treatment for all whiplash victims.

More invasive treatments for whiplash are usually reserved for those who develop chronic symptoms. One of the more effective treatments appears to be percutaneous radiofrequency neurotomy for the treatment of facet joint pain. Lord et al. found that in patients that had facet pain confirmed by double-blinded blocks, neurotomy provided significant pain relief for an average of 263 days, compared with 8 days in the control group (which had the electrode tip placed through the skin onto the joint but had only a 37°C probe instead of the therapeutic 80°C).[76] Similar results have been seen by others.[140,141] Botulinum toxin type A (100U) injected into the splenius capitis, rectus capitis, semispinalis capitis, and trapezius has been shown to significantly improve neck ROM and decreased pain for up to 3 months.[142] It is also significantly reduced headache intensity and frequency in patients with WAD.[143] Interestingly, steroid injection into the facet joints has not been shown to be effective.[144]

SUMMARY

Whiplash is an acute, cervical soft tissue injury, consisting of hyperextension and/or hyperflexion forces, resulting in musculoligamentous sprain or strain. The diagnosis typically excluded fractures or dislocations of the cervical spine, head injury, or alterations in consciousness, and plain radiographs and MRI scans of the cervical spine are typically negative. WAD includes a constellation of symptoms that can also include arm pain and paresthesias, headache, dizziness, visual disturbances, problems with concentration and memory, and psychological illness.

Symptoms that persist beyond 6 months are considered chronic. The true incidence and source of acute and chronic whiplash symptoms is still under investigation but may include the facet joints, intervertebral disks, ligaments, and paraspinal muscles. Acute treatment of stable injuries may benefit from early ROM exercises. Chronic symptoms may benefit from percutaneous radiofrequency neurotomy for the treatment of facet joint pain or botulism injections for myofascial pain and decreased ROM. Exercise and cognitive and behavioral approaches may also be helpful, but more research is needed in this area.

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Cervical Spine Injuries in Athletes

EPIDEMIOLOGY

Sporting events account for 7.5% to 10% of all spinal cord injuries (SCIs), only surpassed by motor vehicle accidents, violence, and falls.[1,2] With a median age of just 24 years, sports-related SCIs rank second when considered in the first three decades of life.[3] Several contact and noncontact sporting activities (including football, ice hockey, rugby, skiing, snowboarding, diving, and equestrian sports) place participants at particularly elevated risk for SCIs.[4–6]

A wide-spectrum of injury severities are recognized and occur at all levels of participation, from high school to professional athletes. Serious injuries with neurologic sequelae fortunately remain infrequent. According to the data compiled by the National Center for Catastrophic Sports Injury Research,[7] there have been a total of 256 cervical SCIs with incomplete recovery since 1977 (with 211, 31, and 9 of these injuries involving high school, college, and professional players, respectively).

Interestingly, these data indicate a reduction in the number of cervical cord injuries (including quadriplegia) when compared with data published in the early 1970s. In football, this reduction is commonly attributed, at least in part, to the 1976 ban on spear tackling (defined as the intentional use of the helmet as the first point of contact with another player), which involves transmission of axial compression forces to the cervical spine in slight flexion.[8] For instance, there were 34 cases of quadriplegia in 1976 and only 5 cases in 1984.[9]

A high index of suspicion combined with an understanding of cervical alignment and architecture and comprehension of the mechanics exerted during a sporting event is imperative to the appropriate management of these injuries.

CLASSIFICATION OF INJURY TYPES

NERVE ROOT OR BRACHIAL PLEXUS INJURIES

Colloquially referred to as "burners" or "stingers," these injuries represent the most common cervical injury. They are manifested by transient loss of function with *unilateral* searing or lancing pain following a collision. A prospective study of college football players identified an incidence of 7.7% per year[10] and an incidence as high as 65% has been suggested over the athlete's career.[11,12]

The player may initially complain of weakness and a radicular burning sensation extending down a dermatomal distribution. These complaints most commonly involve the upper trunk distribution and may last anywhere from a few minutes to several hours. Weakness of shoulder abduction (C5) and elbow flexion (C5, C6) usually remits within 24 hours but may last as long as several weeks.[13] Although function gradually returns from proximal to distal muscle groups, the prognosis is most directly related to whether there is underlying neuropraxia, axonotmesis, neurotmesis, or a combination thereof.

Three primary mechanisms have been implicated: (1) momentary stretch injury to the upper trunk/cords of the brachial plexus caused by the head being driven opposite the depressed shoulder and extended arm (as seen with blocking or tackling in football), (2) compression injury to the cervical nerve root at the neural foramen caused by extension and rotation of the cervical spine toward the painful arm that secondarily stretches the fibrous dentate ligament attachments, or (3) a direct blow in the area of Erb's point (where the upper trunk is fixated to the transverse process).[14] The extension-compression mechanism is usually associated with neck pain and can be reproduced by the Spurling maneuver. Chronic symptoms are more frequent in older athletes in the setting of cervical disk degeneration and are most commonly associated with an extension-compression injury mechanism.[15,16]

Return-to-play is justified if symptoms resolve rapidly and examination reveals full painless neck range of motion with

normal strength. Recurrent episodes or symptoms lasting greater than 24 hours constitute relative contraindications. The most critical treatment obligation of the treating physician is to rule out an unstable cervical injury. Decreased or painful neck motion, neurologic deficit, or evidence of myelopathy mandate that the athlete be withdrawn from play and thoroughly evaluated, including with appropriate imaging studies.

ACUTE CERVICAL SPRAINS AND STRAINS

In these injuries of the paraspinal musculotendinous unit, athletes present with pain localized to the cervical spine following direct jamming of the neck. Examination reveals limited cervical range of motion, but the neurologic findings are otherwise unremarkable, including no evidence of radicular sensory disturbance. Radiographs including flexion-extension views furthermore reveal no abnormalities.

Players are removed from play until full range of motion is restored and the pain, which may be treated with nonsteroidal anti-inflammatory agents, has resolved. As usual, proper immobilization and stabilization is instituted if a fracture or dislocation is encountered. Also, in the event of persistent symptoms, a magnetic resonance imaging (MRI) is necessary to assess for disk herniation.

INTERVERTEBRAL DISK INJURIES

Most commonly, chronic disk changes are seen in athletes involved in contact sports. As many as 32% of freshman football recruits were found to have roentgenographic evidence of either occult fractures, disk space narrowing, or osteophytes/degenerative changes.[17] In these athletes where the cervical spine is exposed to constant loading, MRI findings may commonly reveal a disk bulge with no obvious herniation. Management in these circumstances is conservative with resumption of contact activity delayed until painless full cervical range of motion is seen.

Although much less common, a ruptured cervical disk should be considered whenever there is an acute onset of transient neurologic deficits along with negative radiographs. The clinical symptoms and signs vary from a radiculopathy to an anterior cord syndrome involving acute paralysis of the upper, lower, or all four extremities. Following the diagnosis of an acute disk herniation with neurologic symptoms, either with an MRI or computed tomography (CT) myelogram, surgical treatment may be required. The senior author's preferred approach is an anterior cervical diskectomy and instrumented fusion (Fig. 12-1), although a variety of other approaches exist as reviewed elsewhere.[18]

CERVICAL DISLOCATIONS AND FRACTURES

Cervical dislocations and fractures most commonly occur in collision sports when axial loading forces surpass the yield potential of the vertebral bodies and/or the supporting ligamentous structures.[19,20] Flexion is the primary disruptive force in a smaller percentage of cases. When acutely unstable, these injuries constitute the most common cause of SCIs in collision sports. Cervical spinal instability may be defined as the loss of the ability to limit patterns of displacement under physiologic loads to prevent either any initial/further damage to the spinal cord or nerve roots, major deformity, or incapacitating pain.[21]

Although a wide spectrum of pathologies exists including subluxations, dislocations, and fractures (with or without neurologic damage), the majority of injuries occur in the lower cervical region. Similarly, a range of neurologic dysfunction is observed from partial cord damage with relative preservation of sensory or motor function (with the central cord syndrome being the most common pattern) to complete quadriplegia. The "burning hands" syndrome is a central cord variant with the pathology most likely affecting the medial spinothalamic tracts.[22]

Common severe injury patterns include burst fractures, which stem from severe axial loading (with varying degrees of flexion forces). Extrusion of disk contents and displacement of bone fragments through the fractured endplate result in cervical cord compromise and injury resulting from retropulsion of material into the spinal canal. In contrast, "teardrop" fractures result from severe flexion-axial loading. With compressive deformation of the vertebral body, there is progressive lengthening and failure of the posterior ligamentous complex leading to retropulsion of the posterior body into the spinal canal. CT and MRI scanning is undertaken to evaluate the severity of neural compression and injury, and ligamentous damage. Skeletal traction is often required for realignment and decompression of the canal followed by an open decompressive/stabilization procedure.

Less commonly, flexion-distraction injuries result from either a direct blow to the occiput or rapid deceleration of the torso. These injuries may be unilateral or bilateral and are associated with disruption of supraspinous/interspinous ligaments, the ligamentum flavum, and the facet capsule. Although cervical cord injuries are most commonly associated with bilateral facet dislocations,[5,23] accompanying rotational forces may lead to unilateral dislocations that are also associated with neurologic compromise in approximately 25% of cases[24] (Fig. 12-2).

CERVICAL CORD NEURAPRAXIA

With an incidence in collegiate football players estimated at 1.3 per 10,000 athletes, cervical spinal cord neurapraxia with transient quadriplegia/paresis usually presents as temporary *bilateral* burning paresthesias.[25] Associated motor abnormalities range from bilateral upper and lower extremity weakness to paralysis. Episodes are transient with complete recovery occurring within minutes. However, some patients display residual symptoms for up to 48 hours. Neck pain and loss of
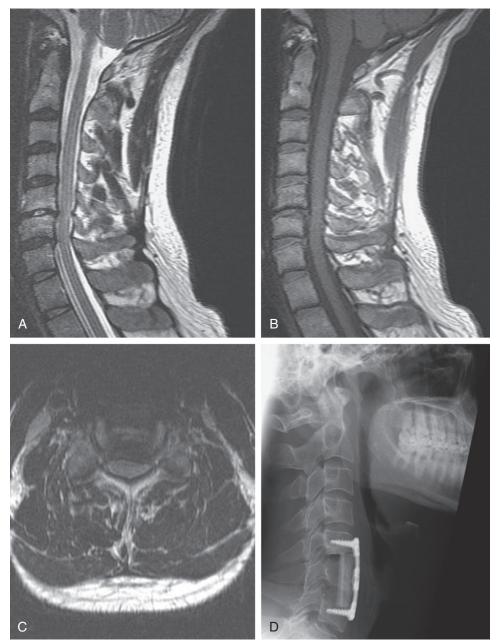
Fig. 12-1 Case of 44-year-old man who sustained a severe hyperflexion of the neck following a fall during mountain biking. He experienced transient quadriplegia followed by complete motor recovery and also developed chronic bilateral painful dysesthesias extending distally from the dorsum of his forearms along a C7 distribution. *A,* Sagittal T2-weighted MRI (1 month following incident) showing multilevel cervical stenosis and cord compression most prominent at the C5-C6 level. Bright T2 and *(B)* low T1 intramedullary signals are most in keeping with an area of myelomalacia. *C,* Axial T2-weighted slice at the C5-C6 level confirms more prominent indentation on the left. *D,* Lateral cervical radiograph following anterior decompression and C6 corpectomy with placement of strut graft and anterior plate.

cervical motion are not typically experienced at the time of injury. Also, these injuries do not predispose athletes to permanent neurologic damage (Table 12-1).

Although radiographs show no evidence of fractures or dislocations, narrowing of the cervical canal has been demonstrated in this population as compared with asymptomatic controls[25] (see Fig. 12-1). Cervical spinal stenosis is defined as a reduction in the anteroposterior diameter of the spinal canal, resulting from congenital, developmental, degenerative, or post-traumatic mechanisms (or a combination thereof). Most commonly, forced hyperextension (but also forced hyperflexion) in a stenotic canal results in momentary
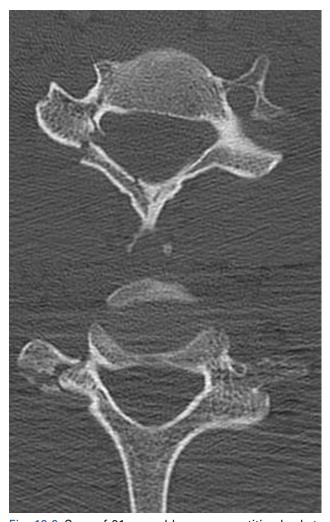
Fig. 12-2 Case of 21-year-old man competitive basketball player who was involved in a diving accident. He did not sustain any neurologic deficit. *Top* and *bottom,* Representative sequential axial CT images showing right C6/C7 laminar fractures extending to the lateral masses. The patient was managed conservatively in a halo vest with no complications.

compression of the spinal cord between the posteroinferior margin of the superior vertebral body and the anterosuperior lamina of the inferior vertebral level.[26]

Wolfe first described measuring the space for the cord on lateral cervical radiographs as the distance from the middle of the posterior surface of the vertebral body to the most anterior point on the spinolaminar line.[27] Because these measurements are often inaccurate because of technical considerations, Torg and Pavlov[28,29] proposed that the width of a given vertebral body divided by the corresponding canal diameter is a more reliable measure of cervical stenosis. They concluded that a ratio of less than 0.8 indicated significant stenosis and constituted a risk factor for neurologic injury in contact sports (with a sensitivity greater than 90%).

However, several subsequent studies identified very low positive predictive values (PPV) in professional football players for this measure in either (1) determining future neurapraxic injuries (PPV = 0.2%)[29] or (2) predicting effacement of the subarachnoid spaces on MRI scans (PPV = 13%), generally considered to be a more functionally relevant measure of spinal stenosis.[30] Given that the vertebral bodies of professional football players in these case series are significantly larger than those of controls, 33% of asymptomatic and 49% of all players, respectively, have abnormally low Torg ratios of less than 0.8.[30,31] These observations thereby preclude any meaningful clinical applicability for this measure in screening athletes for participation in contact sports.

Cantu and colleagues[32–34] have rather emphasized the relationship between the spinal cord diameter and osseous canal width as the most important parameter. Although not as easily quantifiable, this relationship can easily be determined by observing for a cerebrospinal fluid reservoir around the cervical cord on CT myelogram or MRI studies. It remains to be shown whether this subset of athletes, with no observable cerebrospinal fluid around the cord, is indeed at increased risk for injury.

TABLE 12-1   Symptoms and Signs of Brachial Plexus/Root Injuries Versus Cord Neurapraxia

	BRACHIAL PLEXUS/ROOT INJURIES	CORD NEURAPRAXIA
Duration	Transient	Transient
Sensory disturbance	Pain/burning/tingling radiating from neck down *unilateral* shoulder, arm, and hand	Acute bilateral burning pain and tingling associated with loss of sensation in upper and lower extremities
Motor	Possible associated weakness of deltoid, supraspinatus, and biceps	Paresis to complete paralysis involving both upper and lower extremities

RETURN-TO-PLAY CRITERIA
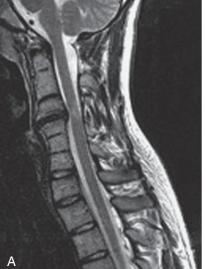
Given the potential catastrophic nature of cervical spine injuries, both the medical and athletic communities have invested much effort over the past several years to identify risk factors and thus define appropriate return-to-play criteria. Unfortunately, although several authors have recommended guidelines,[12,33,35–39] standardized criteria have yet to be widely recognized. Indeed, no consensus on postinjury management was found when more than 100 physicians were surveyed with regard to their personal return-to-play recommendations for 10 case histories.[40] Interestingly, there was also no relationship between return-to-play recommendations and either subspecialty or years in practice. These findings highlight the lack of consensus for predicting injury particularly when decisions are based solely on radiologic evidence.

Once an injury occurs, the consultant physician is often called on to assess whether or not the athlete can return to the playing field. Assessment of risk for further injury involves evaluation of the injury mechanism along with the physical examination and radiographic findings. There is no controversy surrounding the extremes of injury; severely symptomatic athletes (as in central cord syndrome, Brown-Séquard syndrome, or permanent quadriparesis) are excluded from returning to play in contact sports (Fig. 12-3). Athletes with more mild injuries, such as those with "stingers" or transient quadriparesis, and athletes with cervical stenosis must make full recoveries prior to return-to-play.

Asymptomatic athletes with cervical stenosis or athletes who have episodes of cervical cord neurapraxia present the greatest sources of controversy. This debate is particularly heightened in professional sports in which large monetary sums are involved over relatively short careers.

Recurrence rates of neurapraxia in returning football players are estimated to be as high as 56%.[35] Torg and colleagues have outlined a series of guidelines for return-to-play in athletes with congenital cervical stenosis following neurapraxic events.[35,36] They advocate that an asymptomatic athlete with a ratio of less than 0.8 without any other cervical abnormality may return to contact sports without contraindication, whereas a ratio of less than 0.8 and one previous episode of cord neurapraxia represents a relative contraindication. Absolute contraindications include a single neurapraxic event with evidence of cord damage or ligamentous instability, multiple episodes, or episodes with symptoms lasting greater than 36 hours.

Cantu and colleagues have also presented guidelines for return-to-play after transient quadriplegia.[12,33] They proposed that an athlete may return to contact sports following the first episode of transient quadriplegia only if there is complete resolution of symptoms, full range of motion, a normal cervical curvature, and no evidence of stenosis on MRI or myelography. However, relative contraindications include the presence of mild or minimal disk herniations and transient quadriplegia secondary to minimal contact. They further defined "functional" spinal stenosis (i.e., the loss of cerebrospinal fluid surrounding the cord or as
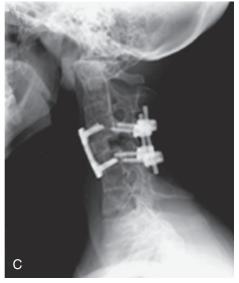
Fig. 12-3 Case of 21-year-old woman with transient quadriplegia following a diving accident. *A,* Sagittal T2-weighted MRI shows multilevel Klippel-Feil anomaly with subluxation at the C4-C5 level associated with a disk herniation, cord compression, and an intramedullary hyperintense T2 signal. *B,* Sagittal STIR sequence indicating likely posterior ligamentous disruption. As flexion-extension films also revealed instability at this level in the context of prior cervical cord injury, the patient required sequential anterior and posterior instrumented stabilization procedures *(C).*

deformation of the cord as documented by MRI or myelography) as an absolute contraindication to return-to-play in contact sports.

Finally, Vaccaro and colleagues[41] have presented an updated and comprehensive series of return-to-play guidelines based on their critical review of the literature. Their recommendations are summarized in Tables 12-2, 12-3, and 12-4.

TABLE 12-2 No Contraindications to Return-to-Play (Slight Risk)

FRACTURES
Healed/nondisplaced C1 or C2 fracture with normal cervical range of motion
Healed subaxial fracture with no sagittal plane deformity
Asymptomatic clay shoveler's (C7 spinous process) fracture

CONGENITAL
Single level Klippel-Feil anomaly (not involving the C0-C1 articulation)
Spina bifida occulta
Asymptomatic with Torg ratio <0.8

DEGENERATIVE/POSTSURGICAL
Cervical disk disease treated successfully with occasional neck stiffness or pain but no change in baseline neurologic status
Following a healed single level anterior fusion (± instrumentation)
Following a single or multilevel posterior cervical microlaminotomy/foraminotomy

OTHER
Up to two prior episodes of a "stinger" (noncomplicated)

TABLE 12-3 Relative Contraindications to Return-to-Play (Moderate Risk)

FRACTURES/DISK INJURIES
Facet or lateral mass fractures
Nondisplaced healed C1 ring or odontoid fractures
Acute lateral disk herniation

DEGENERATIVE/POSTSURGICAL
Following a healed single-level posterior fusion with lateral mass fixation
Following a healed two-level anterior or posterior subaxial fusion with or without instrumentation (excluding posterior lateral mass fixation)
Cervical radiculopathy secondary to a foraminal spur

OTHER
Three or more prior "stingers" or a prolonged symptomatic "stinger" (lasting >24 hr)
A single episode of transient quadriplegia/paresis (noncomplicated)

TABLE 12-4 Absolute Contraindications to Return-to-Play (Extreme Risk)

PRIOR TRANSIENT QUADRIPLEGIA/PARESIS
Two or more episodes of cervical cord neurapraxia
Clinical history or physical examination findings of cervical myelopathy
Persistent cervical discomfort, decreased range of motion, or any evidence of a neurologic deficit from baseline after a cervical spine injury
Presence of intrinsic cervical cord abnormality (on MRI)

FRACTURES/DISK INJURIES
Unstable subaxial fracture-dislocation patterns
Healed subaxial fracture with evidence of sagittal plane deformity
Displaced odontoid fractures
Acute central disk herniation
Residual cord encroachment following a healed stable subaxial spine fracture

POSTSURGICAL
C1-C2 cervical fusion
Following cervical laminectomy
Following a three level anterior or posterior cervical fusion

SOFT TISSUE INJURIES
Asymptomatic ligamentous laxity (>11 degrees of kyphotic deformity or >3.5 mm movement on flexion-extension)
Radiographic evidence of C1-C2 hypermobility with an anterior dens interval of >3.5 mm
Fixed atlantoaxial rotatory abnormality

DEVELOPMENTAL ANOMALIES/COMORBIDITIES
Clinical or radiographic evidence of rheumatoid arthritis
Imaging evidence of a multilevel Klippel-Feil anomaly, basilar invagination, Arnold-Chiari malformation, ankylosing spondylitis, diffuse idiopathic skeletal hyperostosis, a spear-tackler's spine, or occipital-C1 assimilation

MRI, Magnetic resonance imaging.

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Surgical Approaches for the Operative Management of Cervical Spine Fractures

INTRODUCTION AND BACKGROUND

Recent advances in imaging technology, biomechanical research, critical care, and biomedical engineering have revolutionized management of cervical spine injuries.[1–38] Skeletal traction, bed rest, and external fixators[39–43] have been replaced by early open reduction and internal fixation, thereby achieving rapid alignment and/or stabilization aimed toward mitigating secondary insults, promoting neurologic recovery, and preventing late complications.[1,44–57] Adherence to surgical anatomy, the principles of injury mechanism, instability, and choosing the most appropriate technique of internal fixation of cervical spine fractures are fundamental principles that can prevent management complications and ensure better functional outcomes.[58–66] We recognize that it is impossible to fully analyze the mechanism of injury; however, using the mechanistic classification presented by Allen et al.[67] and applying a modern imaging technique would add to the degree of appreciation of vertebral body compression and the degree of posterior ligamentous complex damage (Fig. 13-1).[2,7,19,22–24,26–33,35–37,67–74]

ANTERIOR SURGICAL APPROACH TO THE CERVICAL SPINE

ODONTOID-C1 ARCH RESECTION

Glacial translation of skeletal segments that often is accompanied with pannus at and near the C1-C2 synovial joints, producing anterior compression of the spinal cord,

is not unusual. Under these circumstances, resection of the odontoid peg with the compressive pannus might be necessary to minimize repeated anterior traumatic injury to the spinal cord.[75–77]

CORRELATIVE ANATOMY

The soft palate might need to be incised on one side of the uvula at the median raphe of the posterior pharyngeal wall, including the superior constrictors in the incision. Resection of the anterior arch of the atlas and odontoid process inevitably compromises the transverse ligament, and if a floating decompression is performed, posterior fusion is necessary with or without decompression.

INSTRUMENTATION

Resection of the odontoid process usually is accomplished transorally. For this purpose, either the patient has tracheostomy or transnasal intubation. A Crockard retractor with a tongue depressor is used to expose the oropharynx. Side retractors are used to retract the superior constrictor and expose the C1-C2 region. An operative microscope enters the field from the rostral aspect of the pathologic abnormality or the side of the patient. A high-speed drill is used (cutting burr and diamond) to resect the anterior arch of the atlas and the odontoid process after reflection of the periosteum. An image intensifier is used to verify the position and orientation of the surgical approach.

APPROACH

After tracheostomy and three-point fixation in a slightly extended position are accomplished, a Crockard retractor and tongue depressor are inserted and routine aseptic preparation and drape are performed. A 1-cm incision is made on the left side of the uvula, and a 2-0 silk suture is used to pull this structure superolaterally for better exposure of the oronasopharynx. A longitudinal incision is made in the posterior oropharyngeal wall mucosa in the midline raphe and the entire thickness of the superior constrictor muscle. Bleeders are controlled with bipolar cautery, and side blades of the Crockard retractor are used to deflect the superior constrictor laterally. The anterior longitudinal ligament is cleaned, and a high-speed drill is then

Flex distraction 27

Vertical distraction 4

Distractive ext 8

Flexion 8

Extension 14

Comp flexion 13

Verticle compression 6

Comp extension 7
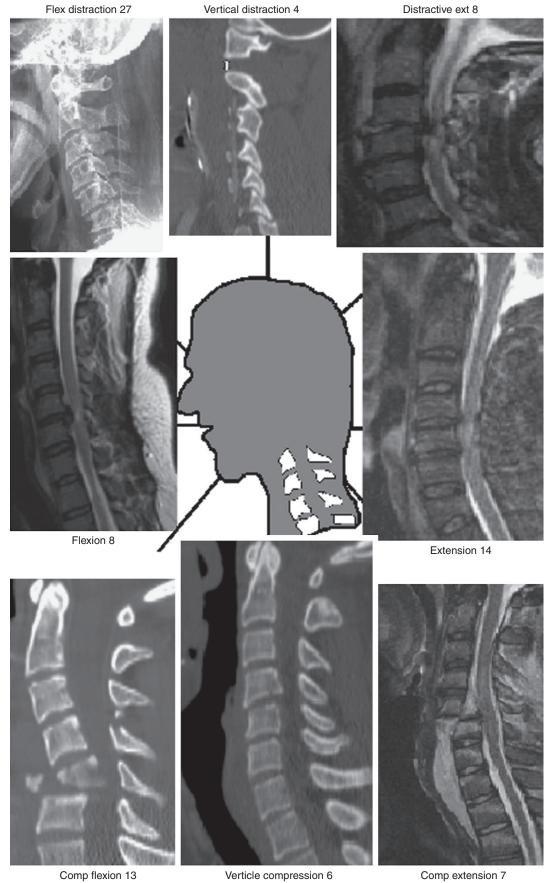
Fig. 13-1, For legend see opposite page.

used to resect the anterior arch of the atlas and the odontoid process. At times, it is impossible to resect the entire mass including the tectorial membrane, and a floating decompression must be performed, leaving a thin layer of soft tissue on the dura to prevent cerebrospinal fluid leakage. After decompression, the superior constrictor muscle and mucosa are closed with absorbable suture. Postoperatively, the patient is kept in a halo vest for second-stage posterior fusion with or without decompression.

ODONTOID FRACTURES

Ten percent to 20% of the more than 6000 cervical spine fractures encountered annually in North America involve the odontoid process.[78,79] A lateral flexion moment plays a significant role in type II odontoid fractures. In 1974, Anderson and D'Alonzo[80] described three types of odontoid fractures with type II comprising two thirds of all odontoid fractures. Although axial fractures commonly are horizontal, anterior and posterior oblique fractures are not uncommon, with the former variety being more prone to nonunion.[78] Odontoid fractures do not usually cause significant neurologic deficit. In an analysis of 2749 patients with neurologic deficits who were admitted to Delaware Valley Regional Spinal Cord Center, Harrop et al.[81] found 126 patients with neurologic injuries at C1-C3; 17 (0.6%) of the injuries were caused by odontoid fractures. On the other hand, among 2347 patients without neurologic deficit, 209 had odontoid fractures.[81] In our series, for the five patients with type II odontoid fractures elected for screw fixation, American Spinal Injury Association (ASIA) scores ranged from 57 to 100. One patient was neurologically intact, and four patients each had traumatic brain injury, central cord syndrome, monoparesis, and quadriparesis. Magnetic resonance imaging (MRI) revealed Class I injury in one patient, Class III injury (Fig. 13-2) in two, and
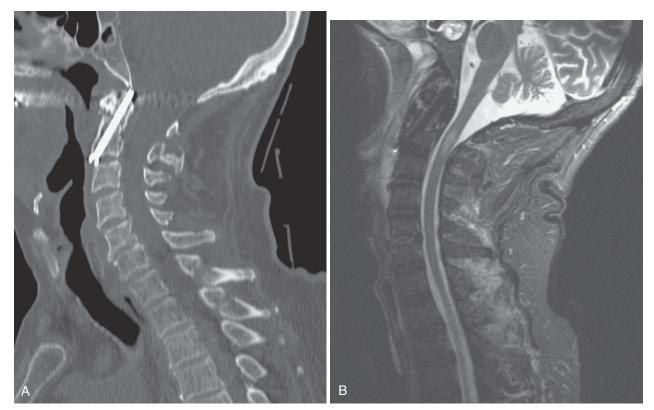
Fig. 13-2 *A,* Midsagittal CT scan of the cervical spine of a 45-year-old man with type II odontoid fracture and partial spinal cord injury obtained immediately after odontoid screw fixation. *B,* Midsagittal T2-weighted magnetic resonance image of the cervical spine of the same patient indicates a high signal change at the level of the odontoid fracture.

normal findings in one.[82,83] The MRI results were missing for one patient. Direct surgical approach to odontoid fractures was first introduced by Nakanishi[84] and Bohler[85] and was rapidly adapted as a sound procedure for internal fixation of type II odontoid fixation while preserving axial rotation of craniocervical joints.[78,79,84–86]

CORRELATIVE ANATOMY

At the level of C3-C4 or C4-C5, which encompasses structures above and below the hyoid bone and thyroid cartilage, the following anatomic structures need to be taken into consideration (Fig. 13- 3)[87–89]:

- Superior laryngeal nerve and its two branches below the hyoid bone; internal laryngeal nerve and external laryngeal nerve; damage to these nerves from the vagus nerve could alter the sensitivity of laryngeal mucosa and cause hoarseness
- Superior thyroid artery inferior to hyoid bone; artery might need to be ligated and divided
- Lingual artery above the hyoid bone; artery must be preserved
- Glossopharyngeal nerve in the vicinity of submandibular gland
- Subhyoid strap muscles; thyrohyoid, sternothyroid, omohyoid, sternohyoid
- Superior constrictor
- Hypoglossal nerve and ansa cervicalis (motor branches of C1, C2, and C3 supplying the subhyoid strap muscles); ansa is anterior to the carotid sheath
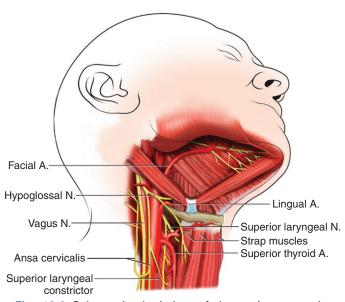
INSTRUMENTATION

- Universal Cannulated Screw System (Medtronic Sofamor Danek, Memphis, TN)
- Shadow-Line retractor system (Cardinal Health, Toronto, Ontario, Canada) also used to expose C2-C3
- Two-plane fluoroscopy is mandatory for proper guidance of the guidewire system
- OSI table (Orthopedic Systems, Inc., Union City, CA) (Figs. 13-4 and 13-5)

CASE SELECTION

Unstable type II odontoid fractures, or those with greater probability of nonunion, are candidates for this surgery. It is unusual, but proper trajectory might fail in patients with extreme barrel chest.

APPROACH

Surgery is performed on the flat part of the OSI table. The exact height of the table is set from the start. Two image intensifiers are placed perpendicular to each other, with one being horizontal and the other vertical above the surgeon's head (see Fig. 13-4). The anteroposterior view machine might need to be turned a few degrees in the sagittal plane to reveal the entire length of the axis. The patient's mouth is kept open by a half roll of Webril (Tyco Healthcare/Kendall, Mansfield, MA), which is placed between the incisors. Two monitors are placed on the left side of the patient immediately in front of the surgeon (see Fig. 13-5). The operator must confirm the feasibility of screw placement before incision; this can be accomplished with the use of fluoroscopy. Accentuated lordosis might be desirable and can be achieved by placing a small role of towels under the patient's neck. Two gel rolls are placed on each side of the head to prevent rotation (Fig. 13-6).

A semitransverse incision line must cover the C3 to C5 vertebral bodies, with the center covering C4. We have

Fig. 13-3 Schematic depiction of the main anatomic structures encountered during upper cervical spine exposure. (Image adapted from *Grant's Atlas of Anatomy,*88 with permission.)

Facial A.
Hypoglossal N.
Vagus N.
Ansa cervicalis
Superior laryngeal constrictor
Lingual A.
Superior laryngeal N.
Strap muscles
Superior thyroid A.

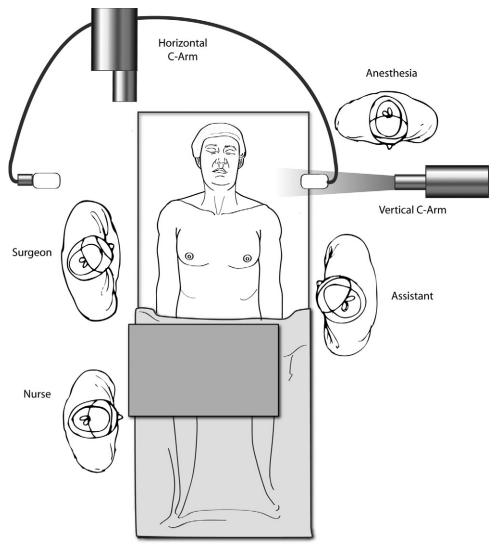Fig. 13-4 Operating room setup for odontoid screw fixation.

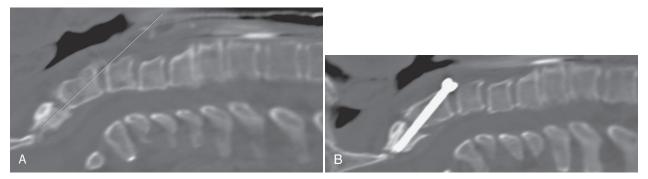Fig. 13-5 Schematic depiction of operating room setup for odontoid screw fixation.

Fig. 13-6 *A* and *B,* Midsagittal CT scans of the cervical spine depict the exact trajectory of guidewire insertion for odontoid screw fixation.

learned that in this way, the surgeon is able to visualize the C2-C3 interspace, remove a small block of C2-C3 disk, and drill a nest in the upper anterior part of C3 for the awl, trocar, guidewire, and tap and then placement of the universal cannulated screw set (see Fig. 13-6). After incision of the platysma, the superficial cervical fascia is sectioned to access the pretracheal fascia. Medial to the carotid pulsation, which is felt by the surgeon, the pretracheal fascia is opened to enter the space between the carotid sheath and ansa on one side and the strap muscles and superior thyroid medially. One should protect the superior laryngeal nerve, superior thyroid artery, lingual artery, and hypoglossal nerve and ansa, all of

which are visible at the level of the hyoid bone. Next, one Cloward retractor (Cloward Instrument Corporation, Honolulu, HI) is used to retract the esophagus medially and the carotid sheath laterally to incise the prevertebral fascia. When the C2-C3 disk is localized, a small block of disk is incised from the midsagittal plane of the C2-C3 interspace. To anchor the trocar of the universal cannulated screw set, a small block of bone is drilled from the anterior body of C3. To prevent anterior screw breakthrough, the surgeon must direct a threaded guidewire at least 2 to 3 mm posterior to the anterior cortex of C2. Introduction of the guidewire is under precise anteroposterior and lateral control of the image intensifier. After introduction of the guidewire, the path is drilled and tapped and a standard or lag screw is then introduced. Although double screws are used at some centers, we think one screw usually is desirable.[78]

C3 TO C7

SUBAXIAL CERVICAL SPINE INJURIES

Spinal cord injury occurs at a rate of 15 to 40 cases per million population worldwide.[81,90] A gender difference seems to be emerging, with older women being involved more often.[91] High-energy vehicular accidents are more likely to produce supra-axial cervical spine injuries.[92,93] Although Hadley et al.,[61] in their *Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries,* recommend three views and supplementary computed tomography (CT) for evaluation of the cervical spine, the introduction of spiral CT and its multiplanar reconstructions have almost entirely eliminated the possibility of missing unstable cervical spine fractures.[2,34,69,74,94–96] At the R Adams Cowley Shock Trauma Center, we include dynamic studies and long-term follow-up evaluation of suspicious looking CT scans to rule out future glacial subluxations.[2,73,97] For evaluation of instability, we depend on the principles clearly mentioned in the literature, which ultimately affect our surgical decision making

regarding anterior versus posterior versus combination fusion.[25,98–103]

We selected 100 patients who underwent surgery from January 2005 through July 2006 to objectively analyze the surgical technique chosen for internal fixation, taking into consideration the mechanistic classification of the cervical spine injury presented by Allen et al.[67] (see Fig. 13-1, Table 13-1). Seventy patients were male, and the mean age was 45.8 ± 18.6 years. Fifty patients had been involved in motor vehicle crashes, 31 in falls, and 14 in recreational sports injuries, specifically shallow dives (nine patients), body surfing (four patients), and wrestling (one patient). Assault and battery were the causes of spinal cord injury in three patients. In two patients, cervical spine trauma was attributed to other causes. Penetrating injuries were not included. Distractive flexion injuries, extension injuries superimposed on spinal stenosis, and compressive flexion injuries were the most frequent injuries encountered in our series. Tetraparesis or tetraplegia and different shades of central cord syndrome were noted in 71 patients. Cervical spine injury was radiologically discovered in 10 patients with traumatic brain injury (Table 13-2). The C5-C6 skeletal segment was most often involved (Fig. 13-7). An anterior approach was used in 41 patients, a posterior approach in 18, and combination surgery in 41 (Fig. 13-8, Table 13-3).

CORRELATIVE ANATOMY

Many of the anatomic structures mentioned in the previous section also apply here, as do the following structures (Fig. 13-9)[87–89]:
- Ansa cervicalis in front of the carotid
- Recurrent laryngeal nerve in the groove between the trachea and esophagus; its course is unpredictable and prone to injury on the right side; many surgeons use the left side approach for C6, C7, and T1 surgical indications
- Inferior thyroid artery at the level of C6-C7 skeletal segments; on the right side, the inferior thyroid artery arises from the thyrocervical trunk and on the left side from the

TABLE **13-1**   Mechanism of Injury in 100 Patients with Traumatic cervical Spinal Cord Injury Admitted to the Shock Trauma Center in Baltimore from 1/2005-7/2006

MECHANISM OF INJURY	
Distractive Flexion Injury Stages 1-4	27
Extension (Spinal Stenosis)	14
Compressive Flexion Injury Stages 1-5	13
Distractive Extension Injury Stages 1-2	8
Flexion (HNP)	8
Compressive Extension Injury Stages 1-5	7
Facet Fracture	6
Vertical Compression Injury Stages 2-3	6
Odontoid Type II	5
Vertical Distraction	4
Hangman's Fracture	2
TOTAL	100

TABLE **13-2** Admission Clinical findings in 100 patients with Traumatic Cervical Spinal Cord injury admitted to the Shock Trauma Center in Baltimore from 1/2005-7/2006.

ADMISSION CLINICAL FINDINGS	
Quadriplegia or Quadriparesis	44
Central Cord Syndrome (CCS)	27
Traumatic Brain Injury (TBI)	10
Incidental Finding on CT	5
Monoparesis	4
Neck Pain	4
Radicular Symptoms	4
Hemiparesis	2
TOTAL	100
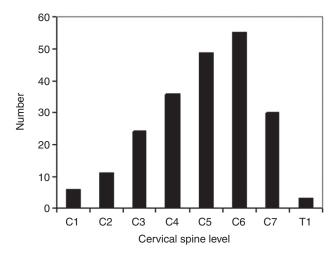
Fig. 13-7 Graph indicates injury distribution of symptomatic cervical spine trauma in 100 patients during an 18-month period at the shock trauma center.

subclavian artery; it is recommended to ligate and divide the inferior thyroid artery when it is stretched and prone to intimal or adventitial damage

- Thoracic duct at the level of C7-T1 on the left side, more prone to injury and causing chylothorax
- Omohyoid muscle must be retracted either inferiorly or superiorly so that the surgeon can have good access to the C6, C7, and T1 skeletal segments; in some instances, especially when more than two skeletal segments are operated on, the omohyoid muscle can be divided and reapproximated with absorbable sutures at the end of surgery
- Apex of the lung might come up to C7-T1 and should be watched for injury

INSTRUMENTATION

- Shadow-Line retractor system
- Cervical curettes, numbers 1 through 5, straight and forward angled
- Kerrison cervical punches 1, 2, and 3 mm, 40-degrees forward angled

- High-speed drill with 3-, 4-, and 5-mm cutter and diamond ball tips
- Microscope microsurgical instrumentation, including Rhoton dissectors, microscissors, and micronerve hooks
- Image intensifier
- Allograft
- Cage system

ANTERIOR APPROACH

At the R Adams Cowley Shock Trauma Center, surgery of acute cervical spine injuries is performed on the Stryker frame (Stryker Corporation, Kalamazoo, MI) or the OSI table. For diskectomy or corpectomy, we use fluoroscopy to localize the skeletal segments of interest. The classic anatomic position of the C3-C4 skeletal segment is at the level of the hyoid bone, C4-C5, thyroid cartilage, and C5-C6 cricoid cartilage. The C6-C7 skeletal segment usually is taken two fingerbreadths above the sternum-clavicle junction. As the number of skeletal segments increases, our incision changes from purely horizontal to slightly vertical. The approach to the prevertebral fascia is as described in the previous segment. One must keep an eye on the inferior thyroid artery at the C6-C7 region and on the left side thoracic duct. One should not violate the most lateral aspect of the longus colli muscles because the sympathetic trunk and related ganglia might be damaged, causing Horner syndrome (see Fig. 13-9).[88,89] Dissection of the longus colli is at the transition of the anterior longitudinal ligament and the muscle. The depth should be at least 7 mm so that the blades of the Shadow-Line retractor can be well anchored. We use two sets of Shadow-Line retractor systems for exposure of more than two skeletal segments (one piggybacking the other). The exposure must be vertical to the trajectory of the microscope light so that the diskectomy or corpectomy can be performed in midline and the fusion plate will not fall to one side, changing the direction of the screws (Fig. 13-10). Part of the diskectomy is performed without magnification, but the posterior annulus and posterior longitudinal ligament with posterior osteophytes are resected by using a high-speed drill and magnification with an operating microscope.
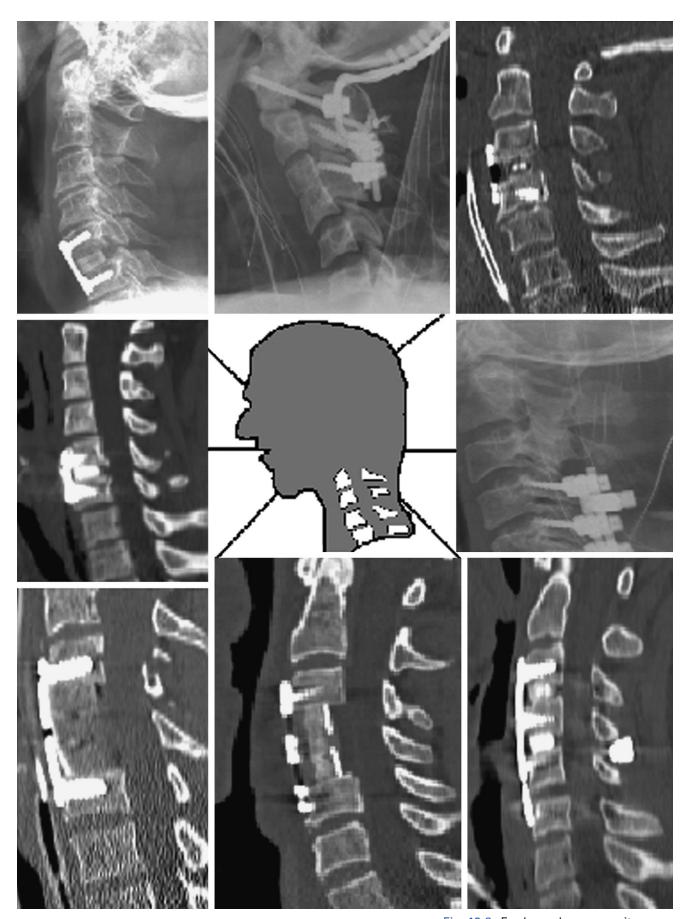
Fig. 13-8, For legend see opposite page.

Fig. 13-8 Clock face composite of plain radiographs and CT scans of the cervical spine indicates postoperative imaging studies of surgical techniques used for specific types of cervical spine injuries as noted in Figure 13-1.

TABLE 13–3   Surgical Management in 100 Patients with Traumatic Cervical Spinal Cord Injury Admitted to the Shock Trauma Center in Baltimore from 1/2005-7/2006

SURGICAL MANAGEMENT	
Anterior Cervical Diskectomy and Fusion (ACDF)	32
ACDF and Posterior Spinal Fusion	27
Corpectomy, Arthrodesis, Plate Fixation and Posterior Spinal Fusion	14
Laminectomy and posterior Spinal Fusion	12
Corpectomy, Arthrodesis, Plate Fixation	5
Odontoid Screw Fixation	4
C1/C2/C3 Fusion	3
Occipito-Cervical Fusion	3
TOTAL	100
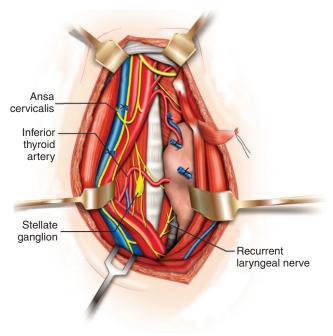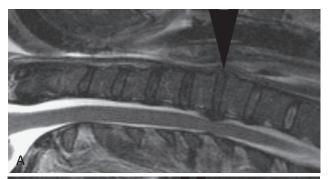
Fig. 13-9 Schematic drawing of the anatomic structures encountered during surgical dissection for exposure of the lower cervical spine. (Figure adapted from the *Atlas of Spinal Operations: Approaches,*87 with permission.)

Fig. 13-10 *A* and *B,* MRI views of a 35-year-old man with traumatic rupture of a C6-C7 disk indicate appropriate vertical trajectory of microscope light to achieve maximal exposure for diskectomy and osteophyte removal.

ANTERIOR CERVICAL DISKECTOMY, ARTHRODESIS, AND PLATE FUSION

Thirty-two patients in our series were treated with diskectomy and fusion. Radiographic injury consisted of traumatic disk herniation in eight patients, distractive flexion in seven, spinal stenosis in six, and distractive extension and fractures of the articulating processes in five. The mechanism of injury was compressive extension in one patient. The level of injury was primarily at C5-C6 in 13 patients, C3-C4 in 11, C6-C7 in 6, and C4-C5 in 2. Anterior cervical diskectomy and fusion involved one vertebral segment in 28 patients, two in 3, and three in 1. MRI revealed Class III injuries in

17 patients, Class I in 3, Class II in 2, and normal findings in 6.[82] The results of MRI were not known for four patients. Anterior cervical diskectomy, arthrodesis, and plate fusion as a stand-alone surgical procedure for traumatic cervical spine injuries are well recorded in the literature.[1,15,104–107]

ANTERIOR CERVICAL DISKECTOMY WITH FUSION AND POSTERIOR SPINAL FUSION

Twenty-seven patients underwent anterior cervical diskectomy and fusion combined with posterior spinal fusion. Injury consisted of distractive flexion Stages 1 through 4 in 15 patients, compressive extension Stages 1 through 5 in six, compressive flexion in two, distractive extension in one, spinal stenosis in one, vertical compression in 1, and facet fracture in one. MRI revealed Class I injuries in 10 patients, Class II in seven, Class III in three, and normal findings in two.[82] MRI results were not known for three patients, and MRI was not performed for two. Injury was at C6-C7 in seven patients, C5-C6 in six, C3-C4 in four, and C4-C5 in four. Multiple levels were involved in seven patients. Injury was complete in 15 patients (ASIA Impairment Scale, Grade A) and incomplete in seven, and four patients had only pain syndrome. One patient had cervical spine injury in association with traumatic brain injury.[22,55,61]

CORPECTOMY AND ANTERIOR STACKABLE CAGE ARTHRODESIS AND PLATE FUSION

Five patients underwent only corpectomy for compressive flexion injury in two and vertical compression in three. All these patients had incomplete injuries (ASIA motor scores 19−95) at C4 to C7 levels. After corpectomy, Poly Ethers Ether Ketone (PEEK) stackable cages and then plate fusion were used (see Fig. 13-8).[7]

CORPECTOMY, ANTERIOR STACKABLE CAGE ARTHRODESIS, AND PLATE FUSION PLUS POSTERIOR ARTHRODESIS AND LATERAL MASS PEDICLE SCREW AND ROD FUSION

In 14 patients, fusion was 360 degrees after corpectomy. Compressive flexion injury was Stages 4 through 5 in eight patients, vertical compression in two, and distractive extension, distractive flexion, and spinal stenosis each in one patient. The level of involvement was C6-C7 in 50% of the patients, and MRI revealed Class I injury in 10 patients, Class II in two, and Class III in one. For one patient, the MRI class was unknown. Eight patients had complete injuries (ASIA Impairment Scale, Grade A), and six had incomplete injuries (see Fig. 13-8).

POSTERIOR SURGICAL APPROACH TO THE CERVICAL SPINE

OCCIPUT, C1, C2

We are saving the lives of a higher proportion of patients with atlanto-occipital and C1-C2 dislocations (see Fig. 13-1). This is in part because of improved care at the scene of the injury and rapid transportation of the victims.[108,109] Traynelis et al.[109] divided atlanto-occipital dislocations into anterior (Type I), longitudinal (Type II), posterior (Type III), and other. Occipitocervical dislocations are diagnosed by using a combination of measurements, CT, and MRI.[109–113] Occipitocervical fusion for atlanto-occipital and atlanto-occipital fusion using newer techniques are preferred, rather than external fixation with halo vest or hard collar (see Fig. 13-8).

POSITIONING

For prone position on the Stryker frame, we use the face mask, and if the Jackson table (Orthopedic Systems, Inc.) is used, either a three-point Mayfield head holder or halo ring can be applied to the main frame.

CORRELATIVE ANATOMY

On each side of the midline, one watches for greater occipital nerve and occipital arteries. Because the incision is in midline, trapezius, semispinalis capitis, semispinalis cervicis, splenius capitis, splenius cervicis, rectus capitis posterior (major and minor), and inferior oblique muscles all fall on each side of the incision.[88,89] In the space between C1 and the foramen magnum laterally, care must be taken at the vertebral artery loop as it exits the foramen transversarium of C1 to penetrate the membrane and become intracranial. The C2 ganglion between the C1 arch and the C2 lamina laterally could be injured during exposure of the C1 lateral mass.

APPROACH TO OCCIPITOCERVICAL FUSION

Certain injuries, such as atlanto-occipital and C1-C2 subluxations, might need occipitocervical fusion. To expose the occiput, C1 arch, and C2 lamina and pars, the following technique is used. An incision is made from the external occipital protuberance to the most caudal portion of the C3 spinous process. Occipital muscles are dissected laterally until the posterior arch of the atlas is reached. The rectus capitis posterior major, inferior oblique, and semispinalis cervicis are dissected free from the spinous process of C2. For C1 lateral mass and C2 pars fusion, the surgeon must expose these two structures. Fluoroscopy helps when drilling the lateral mass of the atlas. Subperiosteal dissection with a straight #4 curette exposes the lateral mass at the inferior surface of the lateral mass of the atlas with very little damage to soft tissues. The C2 ganglion need not be exposed to prevent bleeding from a number of venous plexuses in this region. Retraction of the soft tissues with a Penfield #4 exposes enough of the lateral mass and can be used for creating a pilot hole with a 2-mm high-speed burr. The pilot hole is used for drilling the lateral mass straight down in the sagittal plane and toward the tubercle of C1, as seen on lateral view plain radiographs (Fig. 13-11). A partially threaded screw is used to prevent any unnecessary irritation of the C2 ganglion. Introduction of a pilot hole used to drill the pars of C2 is 5 mm lateral and 5 mm superior to the C2-C3 joint and toward the center of the C2 pedicle, as seen on lateral view radiographs. The length of the screw usually is 16 to 18 mm before reaching
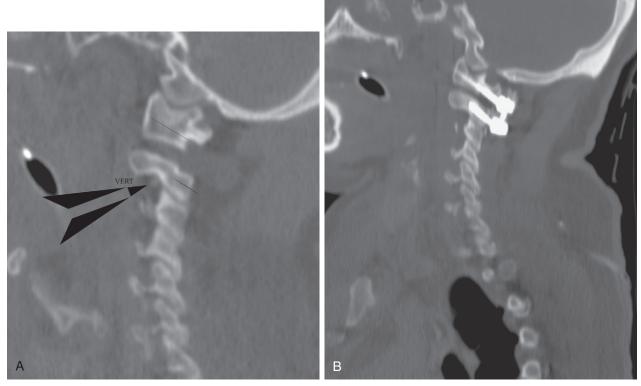
Fig. 13-11 *A* and *B,* CT scans of C1-C2 skeletal segments after a vertical distraction injury (*VERT*) indicate the trajectory of drilling path in C1 lateral mass and C2 pars.

the vertebral foramen in a lateral segment of C2. For three-point fixation, C1 lateral mass-C2 pars fusion usually is supplemented with Sonntag fusion. For that purpose, we isolate the lamina of C2 and the posterior arch of C1 and pass two Songer cables under the arch of the atlas and the lamina of C2; sandwiched in between is a block of iliac crest. Posterior fusion is completed with decortication and arthrodesis of the lateral mass of C1 and the pars lamina of C2 by using local or crest cancellous bone chips.

OCCIPITOCERVICAL AND C1-C2 FUSIONS

Six patients had significant injury to supra-axial bony and ligamentous structures. Four patients had vertical distraction, three involving the C1-C2 joint (two with odontoid fracture) and one involving occiput-C1. One patient had a hangman's fracture with injury to the C3 pedicle. Three patients had occipitocervical and two had C1-C2 Sonntag fusions. One patient had C1-C3 fusion (see Fig. 13-8).

C3 TO C7

LAMINECTOMY
CORRELATIVE ANATOMY

The trapezius, serratus posterior splenius cervicis, and semispinalis cervicis muscles and the supraspinous and interspinous ligaments need to be dissected.

INSTRUMENTATION

Rongeurs, punches, a high-speed drill, microsurgical instruments, and a microscope are used.

APPROACH

Laminectomy is performed either on a Stryker frame, Jackson table, OSI table, or regular operating table. After exposure of the lamina up to the lateral mass junction, spinous processes are removed and the microscope is brought into the operative field. Three-millimeter cutter and diamond drill bits are used to produce two troughs on each side immediately medial to the lateral masses. A 1-mm Kerrison punch is used to free up the laminae on one side by cutting the remaining bone and ligamentum flavum. The laminae are lifted up en block and removed. Adhesions of the inner aspects of the laminae to the dura are sectioned by using a micro-scissor.

LATERAL MASS FUSION
INSTRUMENTATION

Conventional instrumentation for lateral mass screw fixation is used.

APPROACH

Exposure of the lateral masses consists of a continuation of the exposure used for laminectomy. Under fluoroscopic control, lateral masses are drilled. On lateral view fluoroscopic images,

penetration is parallel with the facet articulating processes. The trajectory usually is a fusion of the Roy-Camille and Magerl techniques.[11,14,29,114–117] Before drilling, tapping, and inserting the screws, pilot holes are created by using a high-speed drill equipped with a 2-mm tip (Fig. 13-12).

POSTERIOR SPINAL FUSION WITH OR WITHOUT LAMINECTOMY

Fifty-three patients in our series underwent posterior spinal fusions, 27 of which were performed after diskectomy, 14 of which were performed after corpectomy, and 12 of which were stand-alone with or without laminectomy.

C7 TO T1

PEDICLE

INSTRUMENTATION

The instrumentation was the same as in the previous section for lateral mass screw and rod fixation.

APPROACH

The surgeon must know the exact anatomy of the C7 pedicle. On the axial cuts of the C7 body, the trajectory of the axis of the C7 pedicle is determined. This angle is used to determine the screw path. Under fluoroscopic guidance, we determine the sagittal trajectory of the screw to be parallel with the disk endplates. This might not be enough; therefore, the senior author produces a small laminotomy of C7 near the proximity of the C7 pedicle.[20] The center of the pedicle is approximately 4 mm lateral to the most medial edge of the pedicle (Fig. 13-13).
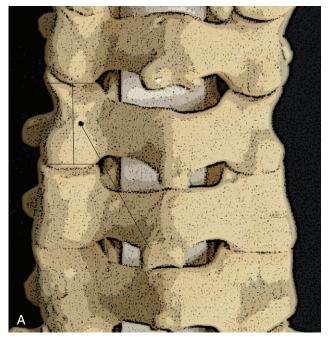
Fig. 13-12 *A* and *B,* CT views of the cervical spine indicate the trajectory of lateral mass drilling and screw insertion.

Fig. 13-13 *A-C,* Plain radiographs and CT scans of C6-C7 skeletal segment indicate the trajectory of C7 pedicle drilling and screw insertion.

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Operative Techniques: Anterior Cervical Decompression, Fusion, and Instrumentation

INTRODUCTION

The anterior approach to the cervical spinal pathology was first described by Robinson and Smith[1] in 1955 for the treatment of disk disease. This approach has been expanded and modified in the years since then to treat various cervical spinal pathologies, including traumatic injury. Cervical spine trauma, when treated surgically, can often be addressed through an anterior approach alone or through a combined anterior-posterior approach. Anterior treatment of the cervical spine offers the advantages of familiarity and satisfactory biomechanical stability for many traumatic injuries.[2,3] This chapter addresses surgery of the sub-axial spine. Transoral procedures and placement of odontoid screws are covered elsewhere in this text.

INDICATIONS OF ANTERIOR CERVICAL APPROACH

Several models of cervical spine trauma have been developed to facilitate decision making in operative repair. The models tend to divide the cervical spine into anterior (the posterior longitudinal ligament and all spinal structures anterior) and posterior (spinal components posterior to the posterior longitudinal ligament) components and classify injury according to location of pathology, mechanism of injury, and neurologic function. No model is universally accepted or describes all injuries adequately, and, therefore no model can provide absolute guidelines for the choice of approach for surgical stabilization of cervical spine trauma.[4–10] The anterior approach is most clearly indicated for pathologies causing anterior compression of the neural elements, such as a herniated cervical disk or a retropulsed bone fragment from a fracture of the vertebral body.[5,11]

When compared with the posterior approach, the advantages of the anterior cervical approach include familiarity to most spinal surgeons, less muscle dissection, avoidance of the risks of turning the patient to a prone position, and greater access to the anterior and middle columns of the spine.[2,6,11] The disadvantages of the anterior approach are an inability to directly address pathology of the posterior elements, risk of injury to important structures in the neck, and at least in laboratory investigations, less rigidity with anterior fixation than posterior.[2,12,13]

For posterior element injury, especially facet subluxation that does not reduce with traction, a posterior route is often selected. However, open reduction may be feasible via the anterior route in many instances, as is explained later in this chapter. Even without subluxation, the anterior approach can be used for posterior element injuries in some cases, particularly with less unstable injuries.[4,6,8,11,14]

For cases in which there is no clear-cut anatomic indication for one approach or the other, both approaches are likely equivalent in terms of clinical results. Of course, both approaches can be used together to achieve greater neural decompression and, with fusion and fixation, greater rigidity.[2,4,8,12,13,15,16] At least one author has recommended a combined approach for fractures in the setting of ankylosing spondylitis, with injuries to the cervicothoracic junction, and in patients with multiple injuries expected to be on prolonged artificial respiration.[2] Furthermore, patients with poor neurologic function as a result of spinal cord injury may require consideration for greater stabilization with a combined approach because of poor neuromuscular control and greater stress on any spinal construct. Greater internal stability decreases the need for a restrictive external orthosis such as a halo vest and permits the initiation of early and aggressive rehabilitation.[4,6]

EVALUATION OF THE PATIENT

Standard evaluation consists of an appropriate history and exam and radiologic studies. A description of the mechanism of trauma is helpful for determining the forces involved in

the injury and the appropriate treatment. A neurologic examination can also help determine the course of treatment and the urgency of any interventions. Adequate documentation of all findings is mandatory.

Patients with multiple traumatic injuries often require stabilization of other injuries prior to addressing cervical spine problems. Patients with major trauma should be assumed to have a spine injury, and thus the spine should be kept in a neutral position with a cervical collar, log rolling, and so forth, until spinal injuries are ruled out. High cervical cord injuries can result in hypotension because of loss of sympathetic function that should be corrected prior to any surgical intervention.

For significant injuries or suspected injuries, imaging studies should include not only plain cervical spine films, but also a thin-cut computed tomography (CT) of the cervical spine with sagittal reconstructions. An injury not seen on initial imaging, such as a purely ligamentous injury in the setting of muscular spasm, or a minor-appearing injury can appear as an unstable injury on follow-up studies at a later date.

A magnetic resonance imaging (MRI) may also be obtained in the stable patient if further soft tissue detail is warranted, which is often the case with an abnormal neurologic examination that localizes to the cervical spine. An MRI is also an option in cases in which cervical spine traction is to be applied, or a purely posterior approach is to be considered for a posterior spinal injury because a disk herniation not seen on CT or x-rays can cause neurologic deterioration in the patient placed in traction. Others have disputed this, however, and recommend immediate closed reduction of cervical spine subluxations in awake patients without first obtaining an MRI.[6,17–21]

SURGICAL ANATOMY

A basic understanding of regional anatomy is necessary for safe surgical access. Provided here is a brief review of important structures encountered during an anterior cervical approach.

Beginning with the anterior neck surface, various landmarks should be visible or palpable. These landmarks aid in placing an incision at an appropriate level. The hyoid bone, the most superior landmark, is approximately at the C3 body level, whereas the thyroid cartilage is closest to C4. The cricoid ring lies opposite C6. The C7-T1 disk space is estimated to be one fingerbreadth above the clavicle. Also, the large C6 anterior tubercle, also known as Chassaignac's tubercle, should be palpable.[22]

Underlying the skin and subcutaneous of fat is the platysma, a thin muscle innervated by the facial nerve. Under the platysma lies the sternocleidomastoid and the remaining superficial anterior neck musculature. The sternal head of the sternocleidomastoid runs from the sternum in the inferior part of the neck and gradually angles laterally as it travels toward the mastoid process of the skull. The omohyoid, which runs at an angle from the scapula to the hyoid bone, crosses the surgical field around the C5 level and may be an obstacle to the approach; this muscle may be cut with no adverse sequela. Under the trachea lies the esophagus, which is muscular but thin. If perforated during surgery, a serious infection may result.[23]

The carotid artery and internal jugular vein lie anterolateral to the spine, underneath the superficial musculature. The vagus nerve travels in the carotid sheath with these structures. An anterior cervical approach is directed medial to these structures, which, for the most part, are kept out of the surgical field.[22,23]

The recurrent laryngeal nerves arise from the vagus and loop into the chest before returning to the neck. On the left side, the recurrent laryngeal nerve loops around the aorta before proceeding on a relatively direct course to the tracheoesophageal groove, where it travels before innervating the muscle. On the right side, the nerve loops around the subclavian artery before ascending to the tracheoesophageal groove. In less than 1% of cases, the inferior recurrent laryngeal nerve on the right is non-recurrent and travels directly to the larynx after branching off from the vagus.[8,24,25] The superior laryngeal nerves are non-recurrent and the external branches of these course with the superior thyroid arteries, which occur above the level of C4.[25,26]

Another cranial nerve that is encountered in high neck dissections is the hypoglossal nerve, which descends near the internal jugular before traveling medially. The marginal mandibular branch of the facial nerve also travels in the high neck and may be injured with retraction against the mandible, resulting in a lower facial palsy.[23]

The longus colli muscles are part of the deep musculature of the neck and are found along the lateral aspect of the cervical vertebral bodies. Sympathetic nerves lie on the surface of the longus colli muscles and may be injured if dissection along these muscles is carried too far laterally.[26,27]

For the cervical spine itself, there are seven cervical vertebrae with intervertebral disks present inferior to each of the vertebral bodies from C2 to C7. The ligaments present in the rest of the spine are seen here as well: anterior longitudinal, posterior longitudinal, yellow, intraspinous, and so forth. The posterior longitudinal ligament is the most important ligament noted on an anterior approach. It is a two-layered structure with the more dorsal layer being thinner but very resilient.[28]

There are eight pairs of cervical nerve roots with C1-C7 exiting above the pedicles of the like numbered vertebral bodies. The C8 nerve roots, however, exit below the C7 pedicles. The nerve roots are located more ventrally than the spinal cord.

The vertebral artery enters the transverse foramen at C6 before traveling superiorly. The other vascular structure to be aware of is the thoracic duct, which drains into the subclavian vein near the junction with the internal jugular vein[29] (Fig. 14-1).
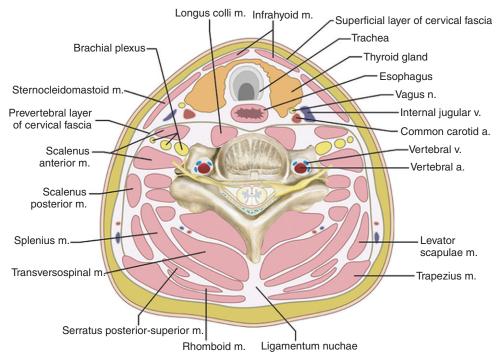
Fig. 14-1 Cross-sectional anatomy of the neck.

OPERATIVE TECHNIQUES

PREOPERATIVE PREPARATION

Once informed consent is obtained, the patient is brought to the operating room. Prophylactic antibiotics are administered approximately one half hour prior to skin incision and sequential compression devices are placed. A bladder catheter is recommended for procedures that are expected to last more than three hours and in patients with impaired bladder function. The patient is intubated and given a general anesthetic. For an unstable cervical spine, which is usually the case in this population, the neck is kept in a neutral position during intubation—this may require the use of an endoscope. An option after successful intubation is the "wake-up" test, in which the patient is allowed to emerge enough to confirm no change in neurologic function.

Once anesthetized, the patient is placed in a supine position on the operating room table with appropriate padding. The neck may be placed in a slightly extended position by using a shoulder roll or dropping the head rest; of course, the decision to do this requires an estimate of spinal stability.

A cervical traction device can be used, especially if it is necessary to maintain spinal reduction during the operation. One type of device simply consists of a chin strap that is pulled in a cranial direction during the procedure when necessary, resulting in extension and distraction. Cranial tongs can also be used. When placing tongs, the skin is cleansed with a povidone-iodine solution, and then the pins of the tongs are placed approximately one to two fingerbreadths above the pinna and one to

two fingerbreadths posterior to the midpoint of the external acoustic meatus. This placement places the neck in some flexion, which is required in most traumatic injuries for reduction; placement approximately one fingerbreadth anterior to the midpoint of the external acoustic meatus provides for extension.[30] The tongs should not be placed in the presence of a skull fracture at the skin entry site. There is a risk of penetrance of the cranial dura with the pins that can result in postoperative cerebrospinal fluid leaks and epidural hematomas, especially if the pins are placed too far anteriorly. Cerebrospinal fluid leaks usually stop with a suture.

The shoulders may be pulled down with wide adhesive tape, or a cloth wrap may be placed loosely around the wrists. The wrap can be pulled by the circulating nurse during the procedure to facilitate radiographic imaging of the lower cervical spine. Excessive traction on the shoulders should be avoided to prevent brachial plexus injuries.[25]

We prefer to use a portable "C-arm" type fluoroscopic device during the procedure. When not in use, the fluoroscopy machine is rolled in a cranial direction to get it out of the way of the surgeon and assistants. This usually requires the anesthesia team to move their equipment further away from the patient than normal.

ELECTROPHYSIOLOGIC MONITORING

Although the use of intraoperative electrophysiologic monitoring for scoliosis correction has been shown to be of benefit in decreasing the likelihood of iatrogenic neurologic deterioration,

its use in spinal trauma is not clear. The most commonly used modality is the recording of sensory evoked potentials (SEPs), which records activity in descending sensory pathways. The theory behind SEP recording is that an insult to the spinal cord at a particular level will usually affect all the pathways traversing that level; therefore, deterioration in SEP measurements will often also indicate a disturbance to motor pathways as well. SEP monitoring requires specialized equipment and a trained neurophysiologist to assess for changes in electrical signal latency and amplitude. Motor-evoked potentials may also be recorded, but there is much less experience with this than with SEP monitoring.[31,32]

TECHNIQUE FOR C2-T1 DECOMPRESSION, FUSION, AND INSTRUMENTATION

Once the patient is properly positioned, the neck, chin, and upper chest are prepped and draped in a sterile fashion. For procedures that span three or fewer disk spaces, a transverse incision is made using the skin surface landmarks or fluoroscopy. The incision begins just beyond the midline and extends to the anterior border of the sternocleidomastoid muscle on the side of approach. A longitudinal incision that approximates the anterior border of the sternocleidomastoid may also be used, especially to expose a longer segment of the cervical spine. We prefer an incision on the left side to minimize the theoretical risks of recurrent laryngeal nerve injury, although no relationship between side of entry and recurrent laryngeal nerve injury has been proven in clinical practice.[24]

After the skin is incised, the platysma is dissected and then divided either in the direction of its fibers or transversely, and then undermined. Veins seen under the platysma are coagulated and cut if they obstruct the approach. The superficial cervical fascia is opened cranially and caudally medial to the sternocleidomastoid muscle. This particular maneuver is key to obtaining adequate exposure of the cervical spine. After the fascia is opened, blunt dissection is carried out medially to the sternocleidomastoid down to the spine. A potential plane is found medial to both the sternocleidomastoid and carotid sheath (which should be palpated) and lateral to the trachea and esophagus. Sharp dissection under the level of the sternocleidomastoid is avoided.[33]

The omohyoid muscle may partially obstruct the approach at around the C5 level. Usually this muscle can be dissected free and moved out of the way, but rarely it will need to be divided and optionally reapproximated near the end of the procedure.

The proper plane will lead directly to the prevertebral fascia of the cervical spine. Often in trauma a hematoma will be present in the area of the fascia or within the fascia itself. This fascia is cleared with a craniocaudal motion using "peanuts." Once this is done, the vertebral bodies and disks should become visible. If unclear, the disks lie at the peaks, and the middle of the bodies are in the valleys of the exposed spine. A spinal needle is placed into a disk space

prior to obtaining a fluoroscopic image to confirm the correct location. The spinal needle may be bent to minimize the chance of the needle tip penetrating too far. If the needle image with fluoroscopy is obscured by the shoulders even after traction is applied, it may be placed in a higher level. Once the correct disk space is confirmed, it is marked with electrocautery after the spinal needle is removed. If adequate exposure is not obtained at this point, it is best to return to a more superficial plane to correct this prior to addressing the spine itself.

At this point, the midline, centered on the midpoint between the longus colli muscles, may be further marked by electrocautery. A good sense of the midline may be obscured if many irregular osteophytes are present but is necessary for placing a cervical plate straight and also to prevent injury to the vertebral arteries.[34] The midline can also be located at the midpoint between the uncovertebral joints. The longus colli muscles are detached medially from the vertebral bodies. This may be done with an instrument such as a Freer elevator or, as we prefer, with monopolar cautery. The electrocautery should be insulated except at its tip to prevent injury to surrounding structures. Minor bleeding will occur as the longus colli are detached. Such bleeding may be controlled by bipolar or monopolar cautery or, occasionally, Gelfoam or bone wax.

After adequate exposure is obtained, cervical retractor blades with teeth are placed under the longus colli muscles, while blunt blades are placed superiorly and inferiorly. The blades help protect both the esophagus and carotid artery, but blades that are not properly seated risk injury to both. The blades should be of proper length; if too short, they have a tendency to displace; if too long, they increase the length of approach to the cervical spine. The endotracheal tube cuff can be deflated and then reinflated after placement of the retractor blades because there is some evidence that this maneuver decreases the risk of recurrent laryngeal nerve injury.[35]

If a subluxation is present, the superior, anteriorly subluxated body may obscure the disk space. In such a case, the anterior inferior edge of the anteriorly displaced body may be removed with a rongeur or drill to allow visualization of the disk.[36]

Distractor pins are then placed in normal vertebral bodies. These pins are placed in the midline at least several millimeters away from the disk endplate. A high-speed drill may be used to penetrate the cortex prior to pin placement. The pins should not be placed in fractured or osteoporotic bone, because as they can break through the bone as distraction forces are applied. Breakthrough may rarely occur with even normal bone.

The standard anterior cervical approach is, in most cases, adequate for exposure of enough of T1 for a C7-T1 diskectomy, C7 corpectomy, and placement of a plate. However, the surgeon should be aware of the increasing distance from the skin to the spine as one descends from C7 and the kyphotic angulation of the thoracic spine. Long retractor blades are often necessary for adequate visualization. Also, lateral

exposure, which is generally not needed in trauma, is limited. The recurrent laryngeal nerve on the right is in an exposed and vulnerable position as it travels from under the subclavian artery to the tracheoesophageal groove; however, this exposed portion is generally below the T3 level. On the left side, it follows a more direct route to the groove after it loops around the aorta.[37] In cases of a nonrecurrent laryngeal nerve on the right, the right-sided approach to the cervicothoracic junction is actually safer because the nerve crosses to the tracheoesophageal groove higher in the neck.[24] The thoracic duct may ascend as high as the level of the C6 vertebral body before descending to empty into the junction of the jugular and subclavian veins on the left, and for this reason, some advocate using a right-sided approach in the lower cervical spine.[37]

For patients with a short neck or when exposure of more thoracic vertebrae is required, the manubrium or sternum proper may be split with the assistance of a thoracic surgeon. Such an exposure would only provide access to the T3 level at most because of the location of the great vessels.[16,37,38] Even with adequate exposure of the C7-T1 disk space, difficulty may be encountered in attempting to place a distractor pin in T1 or visualizing the depths of the disk space because of the angle of the disk space itself and the angle of approach dictated by the intact manubrium. In some respects, a C7 corpectomy is easier than a C7-T1 diskectomy because of the greater visualization provided by the corpectomy. As in the case of subluxation, removing the anterior inferior edge of C7 may improve visualization of the disk space when only a diskectomy is needed.[37]

CERVICAL DISKECTOMY

For a diskectomy, which is performed for a traumatic herniated disk or occasionally for ligamentous instability, the rectangular-shaped incision is made into the disk in question. Of course, the depth of the blade should be controlled and should not penetrate the dura. A pituitary rongeur is then used to remove the initial pieces of disk material.

An operating microscope is then brought in for use. Using a Kerrison rongeur, the inferior lip of the superior vertebral body is removed. This maneuver improves visualization of the disk space and also aids in placement of an interbody graft or fusion device. Distraction with the pins will also improve visualization.

Disk material is removed progressively deeper using a combination of instruments such as angled curettes and pituitary rongeurs. Eventually, the posterior longitudinal ligament will become visible. In most cases of trauma, it is advisable to remove the posterior longitudinal ligament to ensure adequate decompression. If neural decompression is not required, as in pure ligamentous laxity without disk herniation or fractures in the canal, the dissection may end leaving the ligament intact. However, if neural decompression is required, the ligament should be penetrated and removed. This may be done in various ways. We usually penetrate the ligament initially

with an angled curette, nerve hook, or Karlin knife, and then remove the remainder using either a 1- or 2-mm Kerrison rongeur. Posterior osteophytes may also be removed with a Kerrison. Extreme care should be applied with the Kerrison, however, especially in instances of significant stenosis that has not been addressed with a posterior decompression. Removal of osteophytes may sometimes be more safely achieved by drilling the vertebral body endplates until a thin rim of bone is left posteriorly, and then breaking off the remaining bone with a low-profile curette. The neural foramina should be decompressed with Kerrison rongeurs as appropriate.
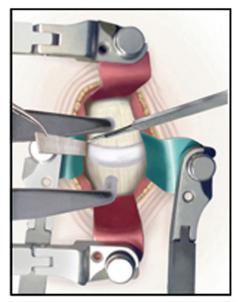
Up to 2.5 cm of disk may be removed safely as long as this width is centered on the midline but usually only 1.5 to 2 cm is necessary for decompression and placement of a graft.[3] Moving too far laterally risks vertebral artery injury. Visualization of the uncovertebral joints aids in identifying the midline (Fig. 14-2).
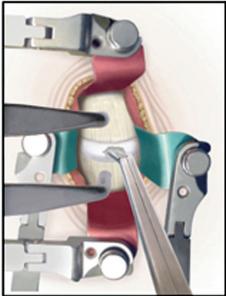
REDUCTION OF SUBLUXATION THROUGH AN ANTERIOR APPROACH

When unilateral or bilateral cervical facet subluxation is present, reduction under fluoroscopy can be attempted through the anterior route after diskectomy. This is performed by placing a lamina spreader or disk space distractor into the disk space and then distracting the two vertebral bodies apart. Traction by an assistant through tongs or by manually pulling on the jaw may be applied simultaneously, often with some flexion of the neck. The surgeon may also directly apply a posteriorly-directed force to the upper vertebral body.[6,8] An alternative method is to apply a corrective force through vertebral body distractor pins.[39] These maneuvers are not always successful, in which case the wound must be closed prior to placing any graft or material, the patient is turned to a prone position, and the subluxation is then addressed through a posterior approach. Then, once the posterior aspect of the procedure is completed, the surgeon must turn his or her attention again to the anterior wound and complete the fusion and fixation.[36] The anterior approach to reduction of subluxed facets is commonly done in lieu of a posterior reduction and stabilization when a herniated disk is seen on a preoperative MRI. If the anterior approach is the preferred option, an MRI need not be performed because any herniated disk at the subluxed level will be removed prior to reduction, but there is the risk of missing a disk fragment behind a vertebral body[19] (Figs. 14-3, 14-4).

GRAFTING AND INSTRUMENTATION AFTER DISKECTOMY

After the decompression is done, the vertebral body endplates are decorticated either with a high-speed drill or a curette. A piece of precut allograft is then placed into the disk
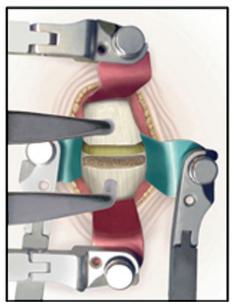
Fig. 14-2 Cervical diskectomy showing placement of retractor blades and a distraction system.

space and slightly countersunk. Alternatives to the allograft include iliac crest autograft and a cervical cage. We prefer cortical allograft filled with demineralized bone matrix in most cases. Autograft has a higher rate of fusion, particularly in multilevel diskectomies (which are rarely performed in trauma), but the risk of donor site complications, such as persistent pain, can be high.[34,40]

Once adequate placement of the graft is achieved, the distractor pins are removed and the holes are filled with bone wax. Cervical plating is used for added stability and decreases the likelihood of graft displacement and postoperative kyphosis.[7] An appropriately-sized plate is selected, contoured if necessary, and secured in place with screws into the vertebral bodies. Fourteen millimeter unicortical screws are used for the average-sized person, 12-mm screws are considered for those of smaller stature.

The wound is then irrigated with antibiotic solution. A final fluoroscopic image may be obtained. The retractor blades are removed and a few moments are spent searching for bleeding points, which are addressed with bipolar cautery. With significant blood oozing, a small drain may be placed. The platysma and dermis are closed with absorbable suture, and the skin is closed with absorbable subcuticular suture. A small dressing is placed.

The majority of the strength of the construct is provided by the cervical plate. A halo is not necessary, but either a soft or hard cervical collar may be used.[28]

CERVICAL CORPECTOMY

A cervical corpectomy builds on the diskectomy. This procedure is performed for unstable fractures of the vertebral bodies. Although some vertebral body fractures may not appear unstable initially, some patients develop a progressive kyphotic deformity that eventually will have to be addressed surgically.[41]

Once exposure is achieved, the distractor pins are placed into the vertebral bodies above and below the ones to be removed. A partial diskectomy is performed at the disk levels above and below the bodies to be removed. A Leksell rongeur is then used to remove the bodies, starting in the midline. The jaws of the Leksell may now be placed into the disk spaces, allowing for fairly rapid bone removal. This bone, as long as it is not involved with tumor, may be saved for fusion material. Bone removal is continued with various rongeurs and a high-speed drill with a cutting burr. The amount of bone removed should be adequate to decompress the spinal canal and wide enough to accept the material chosen for vertebral body replacement. This width is typically less than 18 mm, but, in any case, the width of the bone removed should be less than 2.5 cm to prevent injury to the vertebral arteries.[34,42] The distance between the foramina transversaria at the midcervical spine is approximately 30 mm. The posterior longitudinal ligament again is removed at the surgeon's option to complete the decompression. Brisk bleeding will occur from the bone as it is cut, and this is controlled with
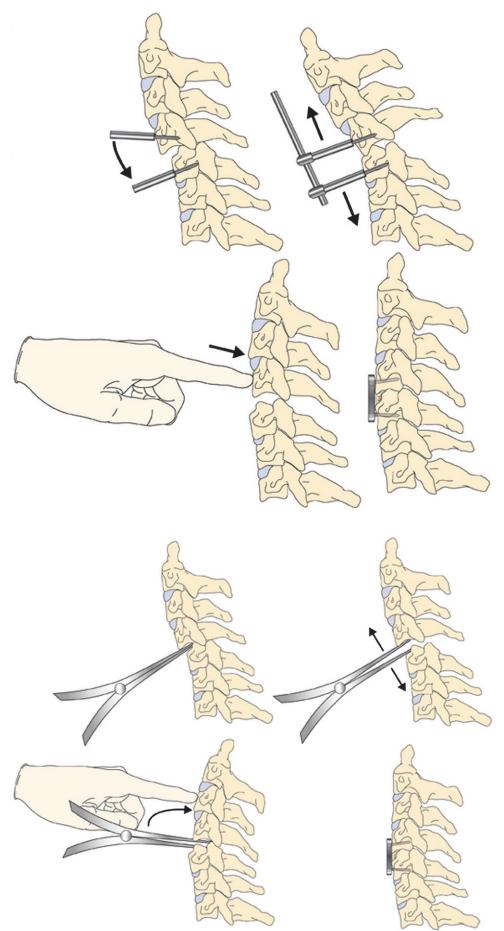
Fig. 14-3 Reduction of cervical subluxation through an anterior approach using distraction posts.

Fig. 14-4 Reduction of cervical subluxation through an anterior approach using a lamina spreader.

bone wax or thrombin-soaked Gelfoam. Bone wax on the endplates is avoided to facilitate fusion.[3]

The vertebral bodies, once removed, may be replaced by a strut graft or titanium mesh cage, either static or expandable. Our current preference for corpectomies of three levels or less is for an expandable cage filled with the corpectomy bone removed earlier. Again the endplates must be decorticated. A thin rim of bone may be left in place posteriorly in both the cephalad and caudal vertebral bodies to minimize the risk of the cage migrating into the spinal canal. The cage is filled with graft material. It is then placed in the void created by the corpectomy and gradually expanded while it is held in place. Expansion is adequate when the teeth on the ends of the cage engage both endplates satisfactorily and the cage cannot be pulled out easily. The locking mechanism is then engaged. After adequate placement, a cervical plate is inserted for added stability. If a static cage is used, the cage is filled with the graft material of choice, the superior end is seated first, and then the inferior end is carefully impacted into place.[42–44]

The wound is closed in the same manner for a cervical diskectomy. Again a drain is considered for persistent oozing, which is more likely to be present in a corpectomy (Figs. 14-5, 14-6, 14-7, 14-8, 14-9, and 14-10).
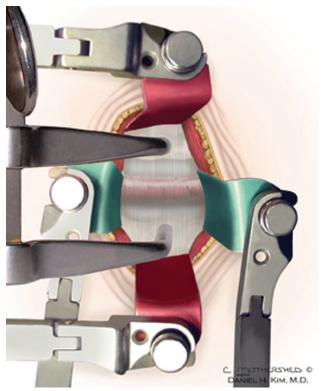
Fig. 14-6 Placement of a distraction system.
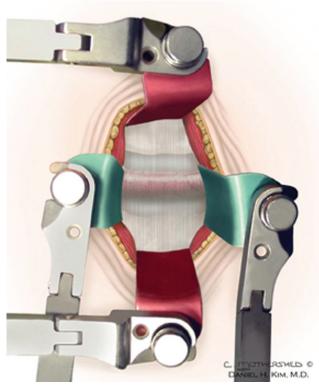
Fig. 14-5 Placement of retractor blades prior to corpectomy for a cervical burst fracture.
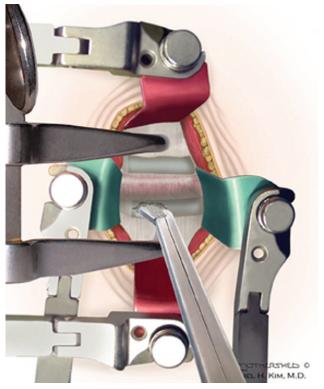
Fig. 14-7 Diskectomy performed above and below the fractured vertebral body.
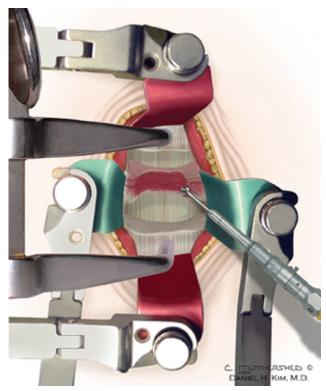
Fig. 14-8 Corpectomy performed using a high-speed drill.

Fig. 14-10 A strut graft is placed.

Fig. 14-9 The posterior longitudinal ligament has been removed and the thecal sac is visible. Further distraction is applied to restore vertebral body height.
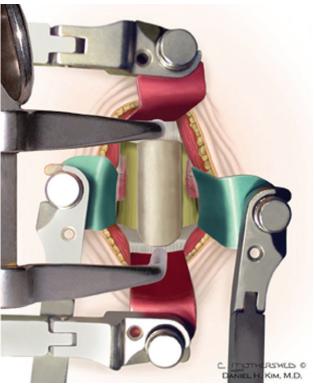
CERVICAL INSTRUMENTATION

There are many different cervical plates available. Self-locking plates with fixed or variable angle unicortical screws are used by most surgeons, and none has proven to be superior to the others.

Modern cervical screws are designed to have only unicortical purchase. Bicortical purchase is still an option, but this offers no significant advantage. For adult patients, the screw length used is usually 12 mm or 14 mm. Fluoroscopic imaging may help in selecting the appropriate length, but we tend to use 14 mm in all adult patients except those of smaller stature.

When a corpectomy is performed, the vertebral body may be replaced by a graft or cage. The graft may be a piece of allograft—either a fibular strut or portion of iliac crest, or autograft, again either a portion of the patient's own fibula or iliac crest. There is a high rate of morbidity associated with iliac crest harvesting.[40,45]

A cage may be static or expandable. A static cage is either premeasured or cut to size intraoperatively. The cage may be straight or have a lordotic curve and comes in various widths. Once the appropriate size is selected or cut, it is filled with the graft material of choice (usually the morselized vertebral body), and the cage is carefully impacted into place.[44–46]

An expandable cage allows for expansion after placement into the space left by the corpectomy. It is gradually

expanded until the ends are embedded into the endplates, at which point it is locked into place by the locking mechanism. The expandable cage may also be filled with the surgeon's choice of graft material.

When a greater than four level corpectomy is performed, a cage is not recommended because of the increased risk of telescoping or pistoning of the device through the vertebral body endplates. For a long segment implant, a fibular strut graft is preferred.[43]

COMPLICATIONS

Potential complications may be divided into three areas: complications related to the soft tissue dissection, spinal decompression, and fusion and instrumentation.

COMPLICATIONS ARISING FROM SOFT TISSUE DISSECTION

Vocal cord movement abnormalities may result from soft tissue dissection. In fact, this is the only complication mentioned in Robinson and Smith's initial brief description of the anterior approach to cervical disk disease.[40] Recurrent laryngeal nerve injury usually results from stretch or compression and leads to dysfunction of the ipsilateral vocal cord and, often, dysphonia. Although the immediate postoperative rate is higher, the rate of permanent hoarseness tends to be less than 4%.[35] There may be a higher rate of recurrent laryngeal nerve injury without dysphonia, as detected by laryngoscopic examination. In addition to dysphonia, unilateral recurrent laryngeal nerve injury can result in aspiration and dysphagia. Bilateral injury is more serious and can result in the additional symptoms of stridor and aphonia.[47] Because in most cases this is a neuropraxic injury, recovery tends to occur over time. If there is no recovery by 6 months to 1 year, the affected vocal cord may be surgically medialized through various methods by an otolaryngologist to reduce the risk of aspiration and improve voice quality.[48] Caution is required when a patient has a history of prior anterior neck surgery. In such cases, the approach may be conducted on the same side as the previous surgery, often requiring navigation through scar tissue, or the approach may be performed on the opposite side after a laryngoscopic examination to ensure proper vocal cord function on the side of the previous surgery.[47]

Carotid artery injury can rarely occur during the dissection. A small laceration may be repaired with 6-0 Prolene suture, but a more serious injury, as may occur with the high-speed drill, may require ligation of the vessel. Obviously, if the collateral cerebrovascular circulation is not adequate, this may lead to a cerebrovascular infarct. Similarly, the internal jugular vein may also be injured, and again this also should be repaired primarily or ligated. Manipulation of the carotid can also result in plaque embolization and stroke.[25]

Potentially the most serious complication arising from the initial phase of the operation is an esophageal or pharyngeal

tear.[49] Such perforations can lead to mediastinitis, meningitis, sepsis, pseudodiverticulum, and fistula formation.[50,51] In the early postoperative period, patients with a tear tend to develop signs of infection. On occasion, a perforation can appear years after the initial surgery, suggesting a late traumatic injury to the esophagus by cervical spine hardware or bone, especially in the setting of a nonunion. Such patients can be asymptomatic but can also complain of dysphagia and recurrent neck pain.[49-51] Plain radiographs may reveal hardware failure, subcutaneous and prevertebral emphysema, and widening of the prevertebral soft tissue.[49,51] Endoscopic techniques and radiographic swallowing studies can help visualize and diagnose esophageal tears.[49–51] A small tear detected during surgery may be treated without primary repair and with intravenous antibiotics. However, in most cases if a small tear is detected during surgery, it is closed with an absorbable suture. Larger tears and chronic or infected perforations should be repaired surgically with the assistance of an otolaryngologist or general surgeon.[50] In addition, consideration must be given to removing or replacing the hardware and graft material. A nasogastric or percutaneous feeding tube is inserted for a period of time to ensure that the tear heals properly after intraoperative closure and intravenous antibiotics are used.[49] Many patients will have some dysphagia after surgery as a result of retraction on the esophagus; such symptoms usually resolve over a few days to weeks without specific treatment.

Other soft tissue injuries that may occur include injury to the trachea, hypoglossal and marginal mandibular branch nerves, and thoracic duct injuries. Injury to the trachea is rare and usually can be repaired directly.[25] The nerve injuries are usually the result of retraction in the high neck and as such are neuropraxic in nature. Although marginal mandibular branch injuries tend to recover, hypoglossal nerve injuries are less likely to heal.[52] A thoracic duct injury can occur in a left-sided approach and is announced by the appearance of chyle in the wound. Treatment requires ligation of the duct.[29]

If occult bleeding occurs into the wound after the procedure is ended, a hematoma may develop. Because there are no firm structures in the anterior neck to tamponade bleeding, a hematoma may become large enough to cause tracheal deviation and airway obstruction.[51] Although most hematomas occur in the immediate postoperative period, occasionally they may occur days later. Such hematomas are optimally evacuated prior to airway compromise. If the airway is already compromised and there is difficulty with intubation, a cricothyroidotomy may be required. Occasionally a patient develops a large hematoma without apparent airway compromise; however, in most cases it is best not to wait to see if the patient runs into trouble. Epidural hematomas can also occur and are announced by neurologic deterioration shortly after surgery. These should be emergently evacuated.[53]

The sympathetic chain may be injured with dissection along the longus colli muscles too far laterally. The chain is

closer to the medial border of the longus colli muscles in the lower cervical spine than in the upper. This will result in Horner's syndrome on that side. Prevention of this injury requires limiting the dissection of the longus colli muscles to their medial edges and ensuring that retractor blades are well seated under the muscles.[25–27]

COMPLICATIONS ARISING FROM SPINAL DECOMPRESSION

During the decompression portion of the procedure, injury may occur to the vertebral artery, especially with a corpectomy. This usually occurs because of working too far laterally with a cutting drill but can also occur with an anomalous artery. Again, a good sense of the midline is necessary to prevent this complication. The preferred method of dealing with a vertebral artery tear is primary repair with 8-0 Prolene, which is usually difficult in the tight confines of the operation. After bleeding is controlled with gentle tamponade, further dissection—including bone removal—is facilitated by retracting the longus colli muscle laterally. Once the artery is exposed, temporary clips are placed and the tear is repaired. If primary closure cannot be completed, the arterial injury should be controlled by tamponade. An injury that cannot be repaired is treated by permanent clipping of the vessel proximal and distal to the injury. This option is used only as a last resort because the sufficiency of the collateral circulation is often not known, although in most cases it is tolerated well.[54] In any case, whether the injury is successfully repaired primarily or packed off, a postoperative angiogram is recommended to assess the need for further intervention. If the tear was not primarily closed during the operation, the vessel should be treated by endovascular means; if this requires sacrifice of the vessel, collateral circulation can be adequately assessed. Not performing an angiogram, particularly in cases in which bleeding was treated with tamponade only, may provide a false sense of security that is shattered when the patient suffers hemorrhage from a pseudoaneurysm or arteriovenous fistula a few days later.[11,46,55]

Dural tears sometimes occur with decompression, and sometimes in trauma a tear is already present but does not become apparent until the decompression is performed. For a simple diskectomy, the exposure is not adequate to allow closure of the dural tear with a suture, and so a dural substitute and fibrin glue may be used. A corpectomy, however, may provide enough working space to place suture and a graft. The surgeon should be careful not to place more fibrin glue than necessary because the glue itself may cause cord compression. A pseudomeningocele after surgery can result in airway compromise from mass effect. Consideration should also be given for a lumbar drain and bedrest for several days postoperatively if a dural tear occurs.[46,56]

Neural elements may be injured during the decompression. Steroids, rehabilitation, and time may help in recovery. The best treatment for this complication is avoidance through careful surgical technique, paying particular attention to neck positioning and the use of surgical instruments near neural elements. Perfect control is required when using a high-speed drill near the spinal cord and nerve roots and when impacting graft material and hardware because the assumption is that surgical technique is the cause of deterioration. However, the true cause is often unclear because in many cases no surgical misadventure is identified.[25,57]

COMPLICATIONS ARISING FROM FUSION AND INSTRUMENTATION

The fusion and instrumentation aspects of the surgery may also lead to problems. A pseudoarthrosis may result, potentially requiring either a revision procedure or a posterior augmentation or both. In cases in which there is no significant instability and the patient is asymptomatic, a pseudoarthrosis may simply be observed. For diskectomies, the rate of fusion decreases with the number of levels and with the age of the patient. Iliac crest autograft results in a higher rate of fusion when compared with allograft, especially when a multilevel diskectomy is involved.[40] However, the rate of morbidity of iliac crest harvesting, which includes chronic pain and fractures, can be high.[40,43]

The hardware may become displaced, especially when the fusion fails. A construct inadequate for the injury may also result in hardware failure. For a cervical plate, failure manifests as screw breakage or pullout. A protruded screw can erode into the esophagus (Fig. 14-11). A cage or strut graft may displace anteriorly (especially if no plate was placed) or posteriorly, occasionally resulting in spinal cord injury.[51] Grafts and cages may also piston into the superior or inferior vertebral bodies, especially in the setting of osteoporosis.[44] Telescoping and subsidence appears to be more of a problem with a fibular strut graft, as opposed to a cage, especially without an anterior plate. On the other hand, the stiffer construct obtained with a cage and plate may place more stress on the spinal elements above and below the construct, leading to adjacent-segment disease and even fractures.[58] Hardware problems usually require surgical revision.[6]

Infection may also result. Luckily, a wound infection rarely occurs in anterior approaches to the subaxial cervical spine. Superficial infections may be treated with oral or intravenous antibiotics and wound care. Deeper infections require treatment with irrigation and debridement and the surgeon should consider the possibility of an esophageal perforation. Except in cases of simply replacing a cervical plate, we usually try to leave any hardware and graft material in place unless instability from fusion failure develops. A closed irrigation system may be helpful in salvaging implants in troublesome infections.
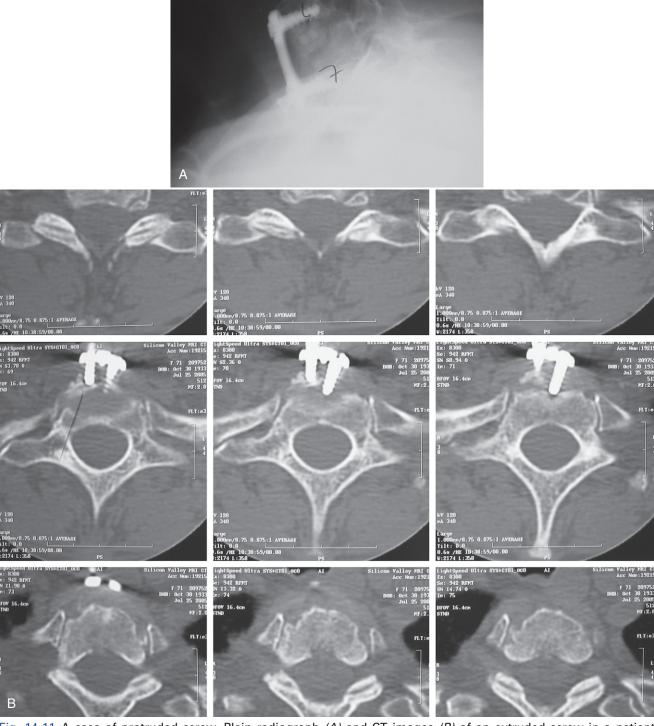
Fig. 14-11 A case of protruded screw. Plain radiograph *(A)* and CT images *(B)* of an extruded screw in a patient treated six months previously for post-traumatic C6-C7 instability at another institution.
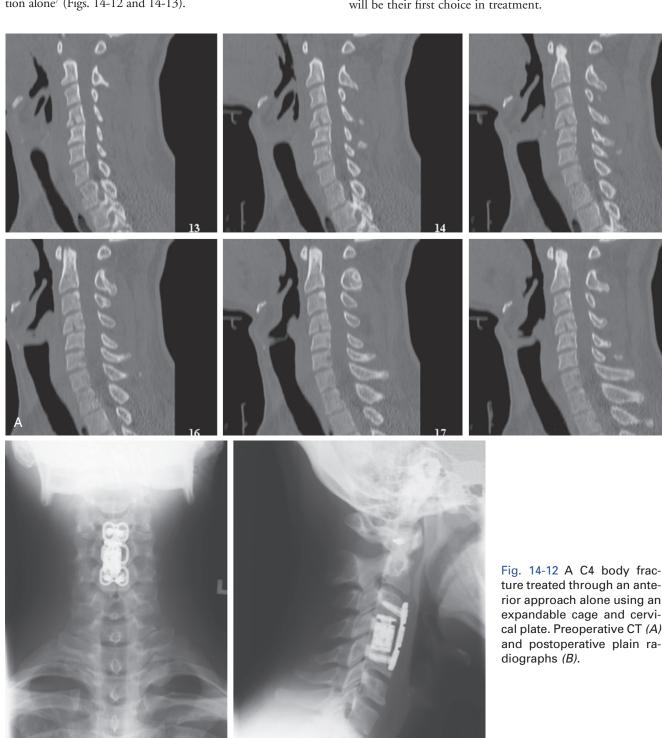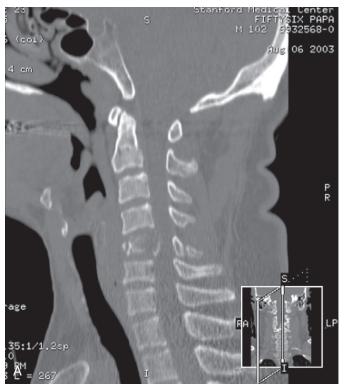
RESULTS

Anterior cervical procedures for traumatic injuries have led to excellent results in terms of fusion and reduction in pain and have also resulted in some improvement in neurologic function.[34] Anterior stabilization results in a smaller incidence of failure of reduction when compared with posterior stabilization alone[7] (Figs. 14-12 and 14-13).

CONCLUSION

An anterior approach to traumatic cervical spine injuries is a straightforward method of dealing with a serious problem. Although debate still continues about what constitutes the best approach for dealing with cervical spine instability, it is safe to say that for most spine surgeons, an anterior approach will be their first choice in treatment.

Fig. 14-12 A C4 body fracture treated through an anterior approach alone using an expandable cage and cervical plate. Preoperative CT *(A)* and postoperative plain radiographs *(B)*.
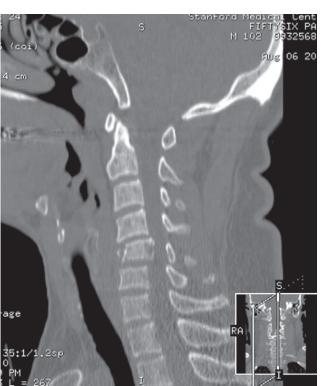
Fig. 14-13 A C5 body fracture with significant anterior and posterior disruption treated with an expandable cage and cervical plate anteriorly and lateral mass screws and rods posteriorly. Preoperative CT *(A)* and postoperative plain radiograph *(B)*.

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Operative Techniques: Posterior Cervical Decompression, Fusion, and Instrumentation

INTRODUCTION

Trauma is a common cause of instability in the cervical spine. However, despite the annual volume of cases worldwide, it has been recognized that there is little uniformity amongs surgeons regarding the most appropriate management and timing of intervention for cervical spine injuries.[1,2] Options available include both nonsurgical and surgical strategies, the latter being composed of anterior approaches, posterior approaches, or combined anterior/posterior approaches. The selection of a treatment algorithm must be individualized for each patient. Factors that must be considered include: mechanism of injury, stability/instability of the cervical spine, neurologic function, patient's overall medical condition and their desires, treating physician's training and experience, and the types of implants available. This chapter focuses on the technical considerations of the posterior subaxial surgical options currently available for the management of acute cervical spine injuries.

PREOPERATIVE ASSESSMENT

The initial management of a trauma patient must include a head-to-toe assessment following the ATLS guidelines to ensure identification and initiate the treatment of any life-threatening or associated secondary injuries. Surgical intervention of the cervical spine should not proceed without a complete and accurate documentation of the patient's neurologic function. The American Spinal Injury Association (ASIA) Scale[3] is a useful tool for the initial neurologic assessment and subsequent follow-up monitoring. A detailed

history of the events leading to the injury, when available, may provide insight into the mechanical forces involved and help direct appropriate clinical decision making. An understanding of the patient's past medical history is also important to the overall patient care. Appropriate blood work should be obtained prior to proceeding to the operating suite. Radiologic investigations should proceed in a systematic fashion. Initial anteroposterior, lateral, and open mouth odontoid views will identify 97% to 99% of injuries.[4] Patients with identified pathology or those with inadequate plain films should undergo further imaging with computed tomography (CT) scanning with sagittal and coronal reconstruction to better delineate the fracture pattern and bony relationships and identify any occult fractures. Dynamic images and magnetic resonance imaging (MRI) scans are obtained when clinically indicated, particularly in the face of neurologic deficit or persisting malalignment requiring open reduction. It should be the intent to obtain the necessary imaging to accurately identify the fracture/injury pattern and plan the optimal intervention.

OPERATIVE CONSIDERATIONS

Appropriate endotracheal intubation may be secured via an awake flexible fiberoptic technique. Preoperatively the anesthetist must be cognizant of the patient's mean arterial pressure, particularly in the setting of spinal cord compromise in which decreased perfusion pressure may worsen neurologic function. The patient's head is then stabilized through pin fixation or a halo ring depending on whether intraoperative traction will be required. If intraoperative monitoring is to be used, baseline readings should be obtained before final positioning. The patient is then turned into the prone position and secured to the operating table ensuring all pressure points are adequately padded. The patient receives appropriate preoperative antibiotics.

POSTERIOR DECOMPRESSION

Traditional laminectomy has been the procedure of choice for posterior decompression in the setting of trauma. The midline is infiltrated with local anesthetic, with epinephrine

and the skin sharply incised. Electrocautery is used to dissect to the level of the spinous processes and the levels confirmed with fluoroscopy. The dissection continues in subperiosteal fashion until the facet joints are identified. This dissection must be undertaken cautiously, particularly in the setting of posterior element fractures, because the dura and/or neural elements are at greater risk. If posterior instrumentation is planned the paraspinal muscles are reflected laterally until the lateral margin of the lateral masses can be identified, careful dissection is essential to ensure that the facet joints maintain their integrity above and below the planned construct. Traditionally the laminectomy proceeded from caudal to rostral over the affected segments using a combination of Leksell and Kerrison rongeurs to remove the lamina and ligamentum flavum. However, with the advancement in high-speed drill technology, placement of instruments within a potentially compromised spinal canal can be avoided. We prefer the latter technique and use the Midas Rex Legend drill (Medtronic, Fort Worth, TX) to create a trough along the medial aspect of the lateral mass at the junction with the lamina. Once repeated on the contralateral side, the interspinous and flaval ligaments are divided so that the lamina(e) may be elevated and removed from the thecal sac without compromising the spinal canal.

Laminoplasty has also been described as a technique for cervical spine decompression but most commonly in the setting of degenerative disease.[5] There has been one report of its use in the setting of traumatic central cord syndrome.[6] The authors reported similar complication rates and neurologic outcomes to laminectomy. However, it should be stressed that none of the patients selected for laminoplasty displayed any evidence of deformity or instability on preoperative imaging. Radiologic evidence of instability would be a contraindication to this approach.

Both of these procedures have the advantage of surgical familiarity and less approach-related complications compared with the anterior approaches. However, they are limited in the degree of decompression that can be achieved, specifically in the setting of a ventral compressive mass such as a bony fragment, herniated disk, or hematoma; and their ability to effectively restore lost cervical lordosis following anterior column disruption. The presence of any of these findings on preoperative imaging should lead the surgeon to consider an anterior or combined approach.

POSTERIOR INSTRUMENTATION

SPINOUS PROCESS WIRING

The management of fractures using posterior wiring as a means for internal fixation was first described in 1891 by Hadra.[7,8] He used a cerclage technique around the spinous processes to stabilize the C5-C6 vertebrae. Rogers[9] and subsequently Bohlmann[10] further modified and expanded the use of this technique.

Both of these approaches require the same exposure described previously for laminectomy, except that the paraspinal musculature must be reflected more laterally to expose the lateral masses. Once the appropriate levels are identified, the posterior elements are decorticated and prepared for grafting. Rogers[9] passed a wire through a hole made in the spinous process at its junction with the lamina at the rostral end of the levels to be fused. The wire was then looped around the spinous process and then threaded through a similar hole in the caudal spinous process to be incorporated in the fusion and tightened in a twisting manner (Fig. 15-1, *A*). Morcellated onlay graft was placed.

Bohlmann[10] preferred to place holes in each spinous process to be included in the construct, made with either a high-speed drill or a perforating towel clip. A wire was passed through both the rostral and caudal levels, similar to Rogers' technique, so that the free ends are on the same side allowing tightening of the wire. Two additional wires are passed through the same holes and then passed through holes placed in unicortical iliac crest grafts. Tightening of these wires causes the grafts to be held firmly to the decorticated posterior elements, one on each side (Fig. 15-1, *B*). The traditional use of stiff 16- or 18-gauge stainless steel wire has now been replaced by softer, multi-stranded, titanium cables, that can be tightened to a measured tension.

The benefits of posterior wiring have recently been retrospectively compared with conservative treatment. In their study, Koivikko et al.[11] showed that patients treated with posterior spinous wiring had a better anatomical outcome and shorter hospital stay compared with those treated nonoperatively. Furthermore, the incidence of late neck pain was statistically higher in the nonoperative group and this correlated with the residual displacement. In addition, there were only six patients in the operative group that required a second procedure, whereas 16 of the nonoperative patients required surgery for progressive deformity or instability. Although technically straightforward and clinically proven, the use of posterior wiring techniques requires intact spinous processes, and therefore had a restricted utility as a technique in the setting of trauma and/or posterior decompression. Moreover, it is well-recognized that the wires can cut through the spinous processes or break, leading to loss of fixation and weakening of the construct.

FACET WIRING

Facet wiring is technically more demanding than spinous process wiring but has the benefit of not requiring intact spinous processes. First described in 1977 by Callahan et al.,[12] the procedure requires exposure and opening of the facet joints to be fused. A hole is drilled through the middle of the inferior articular processes of each facet. The ventral structures are protected with an instrument such as a No. 1 Penfield or a freer elevator. A wire is then passed rostral to caudal through each hole and brought around to secure the bone graft to the dorsal

aspect of the decorticated facet (Fig. 15-1,*C*). Alternatively if the spinous process remains, a hole can be drilled through the center of only the inferior facet. The wire is then passed through this hole and looped around the spinous process of the adjacent lower vertebra (Fig.15-1, *D*). Prior to tightening, the facet joint is decorticated and packed with bone. Unfortunately, the strength of these constructs is suboptimal, and there is a high incidence of the wire cutting through the facet joints, which limits the usefulness of the technique.

LAMINAR CLAMPS

Laminar clamps enable reconstitution of the posterior tension band in the setting of fractures of the spinous process. The Halifax Clamp was first described by Tucker[13] and later by Holness et al.[14] (Fig. 15-2). The laminae are exposed and the insertions of the ligamentum flavum are released from the lamina at the rostral and caudal extent of the construct. The upper and lower hooks of the clamp are sized for length and fit on the laminae. The facet joints are dissected

and decorticated, as are the lateral masses, and packed with bone graft. The clamps should be placed as close to the midline as possible because this is where the spinal canal is most capacious. The construct is tightened and fluoroscopy used to confirm alignment. Further morcelated onlay bone graft is applied. The clamps are good at stabilizing flexion injuries and the placement of an interlaminar bone graft enhances the fusion and limits hyperextension. Similar to spinous process wiring, this technique is limited to use with intact posterior elements. Furthermore, there is considerable concern regarding the potential for canal compromise resulting in neurologic deterioration, particularly in the setting of preexisting stenosis, which limits use in the subaxial spine.

LATERAL MASS FIXATION

Roy-Camille and Saillant[15] first described the use of lateral mass screws and plates to stabilize the cervical spine in the early 1970s. During the 1990s, lateral mass screws gradually took over as the mainstay for posterior internal fixation of the
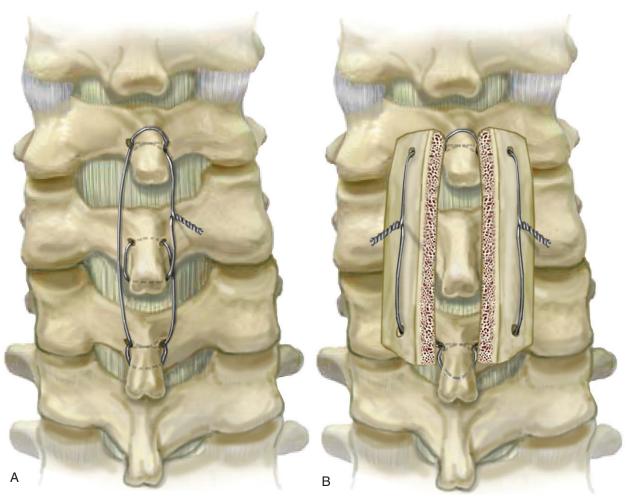

A

B

Fig. 15-1 Illustration depicting various wiring techniques. *A,* Rogers' technique. *B,* Triple-wire technique of Bohlmann.
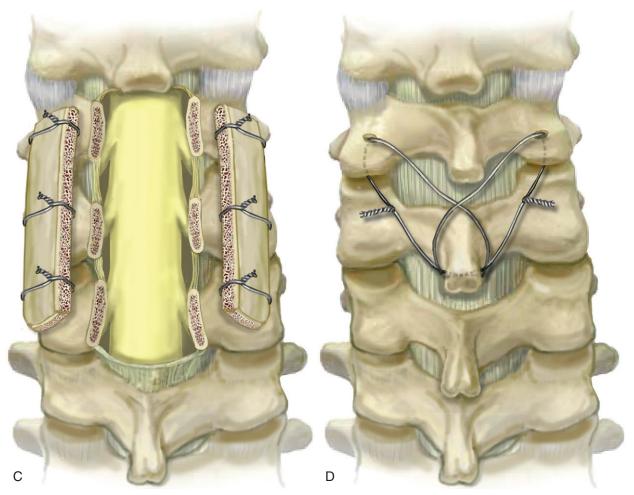
C   D

Fig. 15-1, Cont'd *C,* Facet wiring after laminectomy. *D,* Single level facet and spinous process wiring technique.

Fig. 15-2 Illustration depicting the application of a Halifax clamp.

cervical spine.[16–30] Modifications on the original technique, by Magerl et al.,[18] Anderson et al.,[19] and An et al.,[20] initially targeted different screw trajectories to minimize the risk of injury to the neurovascular structures ventral to the lateral mass (Table 15-1, Fig. 15-3). Pait et al.[31] found that the lateral mass could be divided into "four quadrants." They found

the rostral and lateral quadrant free of ventral neurovascular structures and termed this the "safe quadrant" (Fig. 15-4). Anatomical studies suggest that the An[27] and modified Magerl et al.[32] approaches yield a low incidence of potential injury to the exiting nerve and no risk to the vertebral artery. However, to our knowledge, there are no clinical studies that verify the superiority or safety of one method over another.

A standard posterior approach is used to expose the spine. The lateral margin of the lateral masses denotes the necessary extent of exposure. The levels to be fused are identified and then the rostral, caudal, medial, and lateral borders of each lateral mass are clearly outlined. This allows for accurate identification of the midpoint of the lateral mass, which serves as a reference for the screw entry point. Depending on surgeon preference and local anatomy, an appropriate screw trajectory is chosen and according to the technique of choice, an awl or a high-speed burr is used to penetrate the dorsal cortex in the appropriate starting point (see Table 15-1). A manual or high-speed drill is used to complete the drilling along the appropriate sagittal and axial angles. The depth of the hole can be predetermined from the preoperative imaging but generally a 14- or 16-mm screw should be planned for an

TABLE 15-1   Entry Points and Trajectories for Lateral Mass Screws According
               to Various Authors

TECHNIQUE	ROY-CAMILLE	MAGERL	ANDERSON	AN
Entry point	Midpoint of lateral mass	1 mm medial and 1 mm rostral to midpoint	1 mm medial to midpoint	1 mm medial to midpoint
Lateral angulation	10 degrees	25 degrees	10 degrees	30 degrees
Rostral angulation	0 degrees	30 degrees	30–40 degrees	15 degrees
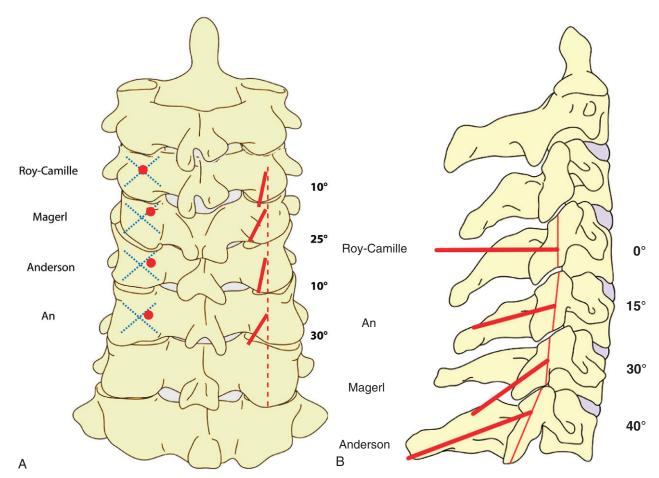
Fig. 15-3 Lateral mass screw trajectories according to Roy-Camille, Magerl, Anderson, and An. *A,* Entry points *(left)* and lateral angulation *(right)*. *B,* Rostral angulation.

average or large adult, respectively. Smaller adult or elderly patients often require 12-mm screws. The pilot hole can be probed with a depth gauge to confirm the ventral cortex is penetrated and the appropriate screw length. The dorsal cortex is tapped prior to insertion of the screw. In elderly or osteopenic patients, consideration should be given to tapping the entire length of the pilot hole. The screws are placed along the same trajectory until "finger-tight," taking care to avoid stripping the screws, particularly in elderly, osteopenic patients. Real-time fluoroscopic imaging or image guidance

software generally is not needed for drilling or screw placement. Radiographic images are obtained at the end of the case to confirm position.

Rigid in design, the prefabricated holes in lateral mass plates do not always correspond to the desired entry point in the lateral masses intervening between the rostral and caudal endpoints. This necessitates the use of different screw placement techniques or, in some cases, suboptimal screw placement, especially in longer constructs. In addition, these plates substantially reduce the dorsal area available for bone
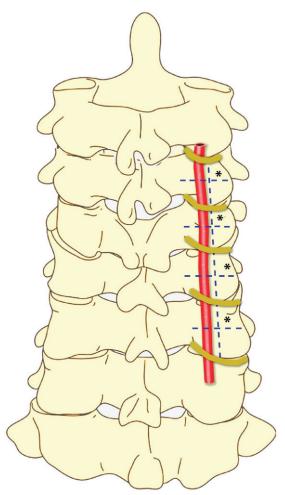
Fig. 15-4 Posterior view of the lateral masses illustrating the four quadrants described by Pait. Approximate locations of the vertebral artery *(red)* and exiting nerve roots *(yellow)* are shown. The * denotes the "safe quadrant."

grafting. These shortcomings have been addressed with the advent of screw-rod systems (Fig. 15-5), which increase the flexibility of screw placement and use either side-loading connectors to attach the rod or polyaxial screw heads that accept the rod in a top loading fashion, thereby leaving space for placement of bone graft. In cases that also require decompression, the holes for the screws may be drilled and tapped prior to laminectomy. This allows the surgeon maximal landmarks for determining the screw entry point and trajectory and also affords the natural protection of the spinal cord during this phase. The laminectomy can then be performed without any obstruction from plates or screw heads followed by the instrumentation and grafting.

Minimally invasive or minimal access surgical techniques have now been applied to the insertion of lateral mass screws. Wang et al.[33] and Fong and du Plessis[34] have recently independently described their use in cervical trauma. The most common use of this technique is to facilitate restoration of

the posterior tension band in patients sustaining bilateral jumped facets after an *ACDF* has been performed. A series of dilators are used to stretch the muscle so that the working tubular retractor can be placed. The use of fluoroscopy is important to ensure that the tubular retractor is inserted along the same trajectory planned for the screw. The margins of the lateral mass are identified and screw placement proceeds in the same fashion described previously. The use of minimal access techniques results in less tissue disruption, thereby minimizing iatrogenic instability and facilitating a faster postoperative recovery. This technique is still in its infancy and has thus far only been used for single-level fusions. Further experience and long-term outcome are needed to ascertain its true benefits.

CERVICAL PEDICLE SCREWS

In an attempt to increase the biomechanical strength of posterior cervical spine constructs, and to address the anatomical challenges of placing lateral mass screws at the C7 level, a number of authors have investigated the use of cervical pedicle screws.[35–39] These studies had suggested that a transpedicular screw has a significantly greater pullout strength than do lateral mass screws, however, there was no significant difference in stability when flexion, extension, lateral bending, or axial rotatory forces were applied.

A number of techniques have been applied to cannulate cervical pedicles. Jeanneret et al.,[40] based on their anatomic work, found that the screw should enter 3 mm caudal to the inferior facet in the midline of the articular mass. The screw should be angled medially at 45 degrees and aimed toward the cranial third of the vertebral body on fluoroscopy. Jones et al.[37] confirmed this trajectory but noted that the angle varied slightly ranging from 38 degrees at C3 to 59 degrees at C7. It is therefore suggested that preoperative CT scanning be used to individualize the trajectory for each patient. Miller et al.[41] also advocate creating a laminar "window" to better identify the pedicle and reduce the incidence of significant breeches. Abumi and coworkers[35] first described the technique and use a combination of anatomic landmarks and fluoroscopy to cannulate the pedicle. Once the articulating masses of the levels to be fused are identified, a burr is used to remove the dorsal cortex posterior to the superior facet and then directly identify the introitus of the pedicle. This is usually marked by bleeding cancellous bone. Under fluoroscopy, the pedicle is probed and the screw placed with a 30- to 40-degree medial angulation to a predetermined depth (Fig. 15-6). The screws are then typically connected using a rod construct and the surrounding bone decorticated and bone graft laid in place.

We prefer to use a combination of techniques. The preoperative CT scans are used to determine the appropriate trajectory angles individualized for each level and side, as well as the screw diameter and length. Once exposed, we perform a small laminotomy at the superior aspect of the lamina so that the pedicle can be directly palpated to help with our trajectory
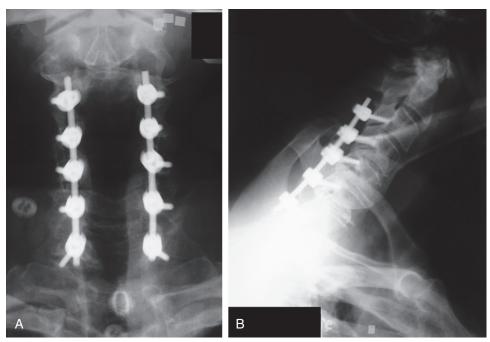
Fig. 15-5 Postoperative *(A)* antero-posterior and *(B)* lateral radiographs illustrating lateral mass screw fixation.
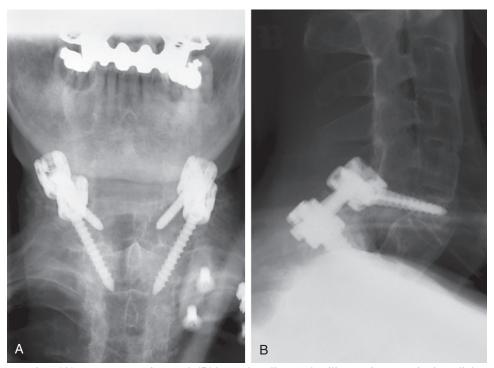

Fig. 15-6 Postoperative *(A)* anteroposterior and *(B)* lateral radiographs illustrating cervical pedicle screws inserted into C6 and T1 in a patient with ankylosing spondylitis. Note the medial angulation of the screws on the anteroposterior (AP) image.

and to confirm there is no medial pedicle breech on insertion. Removing the dorsal cortex and visualizing the entrance to the pedicle determines our entry point.

Although cervical pedicle screws were initially used at the C2 and C7 levels,[42–44] because the pedicles are more capacious,[37,45,46] many authors have suggested that mid-cervical levels can also be cannulated but the risk of misplaced screws (Fig. 15-7) increases if the preoperative imaging suggests the pedicles are less than 4.5 mm in width.[37,44,47] This is reflected in the published rates of pedicle breaches (up to 30%)[44,47] and associated neurovascular complications (3% to 8%).[47,48] These complication rates, to some degree, reflect the technically
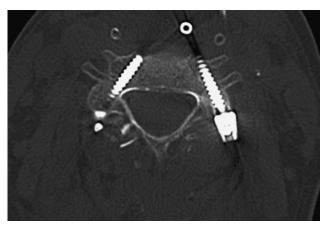
Fig. 15-7 Postoperative axial CT image. The pedicle screw on the right slightly breeches the pedicle. The left-sided screw, however, completely traverses the transverse foramina.

challenging nature and the significant learning curve associated with this technique.[30,47,49]

CONCLUSION

There now are a number of posterior surgical options for internal stabilization of traumatic cervical spine injuries. The posterior approach is familiar to most surgeons and maintains a low complication rate. The wiring techniques have generally been replaced by screw fixation. Because of the greater flexibility with insertion, the screw/rods systems are used more commonly than the screw/plate systems. These various approaches each have their own inherent indications and pros and cons. It is imperative that surgeons are familiar with each of these techniques so that they can optimally individualize treatment for each patient.

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C H A P T E R

16

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ARNOLD H. MENEZES

Pediatric Craniocervical Trauma

INTRODUCTION

Although cervical spine injury is relatively uncommon in children, the craniovertebral junction is the most commonly injured region in young children,[1–6] with some series reporting involvement of the occipitoatlantoaxial complex in nearly 90%.[7] These injuries often are ligamentous disruptions rather than true fractures, resulting in craniovertebral dislocations that require rapid diagnosis for optimal treatment.[8,9]

DEVELOPMENTAL ANATOMY AND BIOMECHANICS

Configuration of the upper cervical vertebrae changes dramatically from infancy through early childhood, reaching an adult form by the age of 8 years.[6,10–12] Several intrinsic and extrinsic factors of the pediatric spine predispose the child to spinal cord and ligamentous injury at more rostral levels because of immaturity simultaneously decreasing the overall incidence of spinal column fracture compared with adolescents and adults.[3,6,11,13–16]

A prevalence of atlas and axis injuries in infants and young children is related to the developmental anatomy of the upper cervical region.[3,10,17–20] The child's head is proportionately larger compared with the body, which results in a higher torque being applied to the neck with acceleration and deceleration stresses, compared with the adult.[21] The fulcrum of flexion and extension is shifted to the C2-C3 level from the low cervical level found in adults. The stabilizing ligaments of the pediatric spine have more laxity compared with the adult spine, and the paraspinous muscles do not reach full maturity until puberty.[10,16,20,22]

Both the occipitoatlantal and atlantoaxial articulations are involved in flexion and extension. The average range of this motion of the occipitoatlantal joint is 13 to 15 degrees, whereas an additional 10 degrees of motion occurs at the atlantoaxial articulation. Of the ligaments providing occiput-C2

stability and limiting excessive movement, the tectorial membrane plays a crucial role, strapping the body of C2 firmly to the clivus and anterior rim of the foramen magnum; sectioning of this ligament in cadaveric studies has resulted in instability with flexion, extension, and distraction.[21,23] Rotational stability is conferred by the alar ligaments arising from the dens to the medial occipital condyle bilaterally,[24] whereas translational stability between C1 and C2 is provided by the transverse ligament, keeping the dens closely approximated to the anterior atlas.

The atlantoaxial joint provides an optimal design to permit rotation. Maximum rotation of the atlantoaxial joint is approximately 45 degrees.[25–27] When this is exceeded, an interlocking takes place between the inferior facet of the atlas vertebra over the superior articular facet of the axis vertebra. If a deficiency of the transverse ligament is present, the anterior atlas arch will sublux forward, producing a unilateral dislocation, and the facet will interlock at a rotation of less than 45 degrees; conversely, an intact transverse ligament will cause no subluxation between the odontoid process and the anterior arch of the atlas.[28–30]

Rotation at the atlantoaxial joint of more than 30 degrees produces an angulation of the contralateral vertebral artery.[19] With greater rotation, stretching of the vertebral artery increases, and at 45 degrees, the ipsilateral artery might have angulation and occlusion, explaining some neurologic deficits from cervical traction, wrestling, chiropractic manipulations, and football injuries.[31]

Only half of the total lateral rotation motion takes place at the atlantoaxial joint[21,22]; the remainder occurs at the subaxial level. The muscular contractions and the muscle tone produce a compression force across cervical motion segments.[26,32,33] The initial axial twist produces a threshold value that overcomes the "interlocking stiffening" of the subaxial segments, allowing for the completion of the rotation to 90 degrees.[32]

Translation and rotation occurring simultaneously is called *coupling*.[34] This occurs at the atlantoaxial joint because of the geometry of the articular surfaces. With axial rotation of the atlas around the axis vertebra, an associated upward movement of the dens occurs in relation to the atlas. Thus, in rotary luxation of the atlas beyond 35 to 40 degrees, a

descent of the cervicomedullary junction seems to occur.[28,30,32] The normal distance from the basion to the odontoid process is approximately 5 mm.[33] Normal translation between the clivus and the anterior arch of the atlas should be no more than 1 to 2 mm.

In a study of the normal pediatric cervical spine presented by Cattell and Filtzer,[35] the predental space between the anterior arch of the atlas and the odontoid process was found to be 3 to 5 mm for ages younger than 16 years. Similarly, ligamentous laxity could explain their finding of an overriding superior arch of the C1 on the odontoid process in extension in 20% of these children. The neuro-central synchondrosis of the axis was present in 100% of children younger than 3 years, in 50% between the ages of 3 and 6 years, and in extremely few older than 8 years. A pseudosubluxation between C2 and C3 was observed in 45% of these normal young children and is attributed to the anterior wedging of the vertebral bodies, poorly developed Luschka joints, ligamentous laxity, and the relative horizontal plane of the articular processes.[17,34–38] These also contribute to instability under applied flexion and rotation stresses.[34,39,40]

CAUSES OF CRANIOVERTEBRAL INJURY

Motor vehicle accidents are by far the most common cause of spinal cord and spinal column injuries in the pediatric population, representing 20% to 72% of cases.[3,6,12,41–43] Pedestrian/auto accidents are particularly common in the infant and young pediatric age group.[7,13,14] The upper cervical spine, especially the craniovertebral junction, is predominantly affected, with mortality rates reported to be from 50% to nearly 100% secondary to atlanto-occipital dislocations (AODs).[41,44] In a recent series of 30 pediatric trauma cases reported by Wang et al.,[44] the craniovertebral injuries in all eight children occurred secondary to motor vehicle-related trauma. In a series of 103 cases, Brown et al.[41] reported that 68% of the patients sustained injuries to C1-C4, 20% of which were dislocations at C1 and resulted from pedestrian/auto accidents.

Upper cervical spine fractures commonly occur in older children as a result of falls, diving, or various sporting activities, accounting for 10% to 15% of spinal column injuries in most series, with some authors reporting 32%.[12–14,45–47]

CLINICAL PRESENTATION OF NEUROLOGIC DEFICITS

The incidence of neurologic deficits from upper cervical spinal injury in the pediatric age group varies widely, exceeding 50% in some series.[7,12,44,45,47] Fatalities from traumatic occipitoatlantal dislocation can approach 100%.[44] Nonfatal neurologic deficits can be complete or incomplete, with incomplete injuries presenting as several varying syndromes. Kewalramani et al.[13,14] and Burke[48] found a strong association between pediatric spine injury and complete neurologic deficits with 69% and 95% of their patients, respectively, experiencing complete deficits. However, the incidence rates of complete injuries reported by Anderson and Schutt[49] and Ruge et al.[12] were less than 20%.

IMAGING TECHNIQUES

Plain radiographs are the first images obtained when upper cervical spinal trauma is suspected. Anteroposterior cranial views, the Townes view, and a lateral view of the skull are necessary. It is imperative to visualize the odontoid process and atlas vertebra using the open-mouth view because an inadequate examination can lead to missed fractures. Criteria used to detect instability at the craniovertebral junction include the following:

1. Predental space of more than 5 mm in patients who are 8 years or younger and more than 3 mm in patients who are older than 8 years
2. Separation of atlas lateral masses of more than 7 mm on the open-mouth view suggests a Jefferson fracture and the possibility of disruption of the transverse portion of the cruciate ligament.
3. Vertical clivus odontoid translation of more than 2 mm indicates occipitoatlantal ligamentous injury.
4. The gap between the occipital condyles and the atlas facets should be minimal so that the occipital condyles are never visible on their own. "Bare" occipital condyles indicate an occipitocervical dislocation.
5. Any abnormal relationship between the spinal canal and foramen magnum is pathologic except for widening of the interspace between the occiput-C1 and C1-C2 posteriorly.
6. Abnormal craniocervical motion dynamics

Considering the high incidence of ligamentous injury, obtaining flexion-extension views should be considered with suspected spinal injury. Areas of suspicion can be evaluated further by using computed tomography (CT) to define the presence of fractures, which often are difficult to visualize on plain films, particularly when they involve the facets or posterior elements. CT reconstructions are also helpful for detecting fractures, assessing facet joint integrity, and determining alignment.[50] The landmarks used in the radiographic criteria of craniocervical instability in the adult, however, might be difficult to visualize or might be morphologically different in young children, making the diagnosis difficult. To that end, and to fully evaluate the spinal cord and subarachnoid space, magnetic resonance imaging (MRI) is the mainstay. With MRI, traumatic epidural hematomas, traumatic disk herniation, and ligamentous injury can be easily documented. MRI has to be used in conjunction with CT when osseous anatomy must be depicted in fine detail.

SPECIFIC INJURIES OF THE CRANIOVERTEBRAL JUNCTION: CLINICAL PRESENTATION AND MANAGEMENT

This transition zone between skull and vertebrae is comprised of the occipital bone, the atlas, the axis vertebrae, and their associated ligaments, the complex interactions of which allow extensive movement yet suggest that it functions as a complex unit. The atlas serves as a washer or bearing between the occipital condyles and the axis vertebra. The diagnosis and treatment of trauma to this region require a thorough understanding of the osseous anatomy, ligamentous structures, and their functional properties together with joint kinematics,[51] as discussed in the next section.

OCCIPITAL CONDYLE FRACTURES

The most common clinical feature of an occipital condyle fracture is loss of consciousness or cranial nerve damage. This injury often is overlooked and is not obvious on plain radiographs but is easily confirmed by CT with reconstructions[31,52–54] (Fig. 16-1). Treatment is most often adequately rendered conservatively with Philadelphia or Miami J collar immobilization.

JEFFERSON FRACTURE

The Jefferson fracture is caused by excessive axial loading, which results in divergent lines of force passing through the lateral masses of the atlas vertebra. The lateral atlantal masses are wedge shaped and thus can be easily displaced in an outward manner, with bursting of the C1 ring at the vertebral artery groove, which is its weakest point. It has been said that atlas fractures are uncommon in children, but this is not the case.[31,55,56] Posterior arch fractures are the most common. However, the anterior C1 arch fracture might occur with axial loading in extreme flexion (Fig. 16-2, *A*). As reported by Levine and Edwards,[57] this type of fracture was described as a four-part burst fracture by Jefferson. In most instances, the Jefferson fracture is considered to be a stable injury that responds well to conservative management with Philadelphia collar immobilization. In instances with combination fractures, halo immobilization might be required. Please refer to Figures 16-2, *B* to *D,* for computed tomographic scans and magnetic resonance images of significant combination fracture and ligamentous injuries at the occipitoatlantoaxial level.

FRACTURES OF THE ODONTOID PROCESS

Odontoid fractures in children are avulsion fractures at the neurocentral synchondrosis, particularly in children younger than 8 years. In addition, ligamentous laxity and incomplete

Fig. 16- 1 *A,* Three-dimensional computed tomographic scan shows an occipital condyle fracture. *B,* Axial view computed tomographic scan obtained through the plane of the occipital condyle shows the parasagittal oriented fracture on the right.

ossification in young children make them more prone to this sort of injury. Both flexion and extension injury can produce odontoid fractures, and because open-mouth odontoid views might be difficult to obtain in children, CT is helpful.[50,58] The treatment comprises realignment and complete immobilization for 8 to 10 weeks in a halo vest or Minerva cast[3,47,59] (Fig. 16-3). Inadequately treated injuries can lead to os odontoideum with chronic instability. In patients older than 8 years, fractures can be classified according to the schema proposed by Anderson and D'Alonzo,[60] as used in the adult: types I, II, or III. Type I fracture has an oblique line that goes through the upper one-third of the odontoid process. This represents an avulsion fracture where the alar ligament attaches. Type II fracture occurs at the lower third of the odontoid at the junction of the odontoid process and the body of C2. The type III fracture extends down into the cancellous portion of the body of the axis, and the
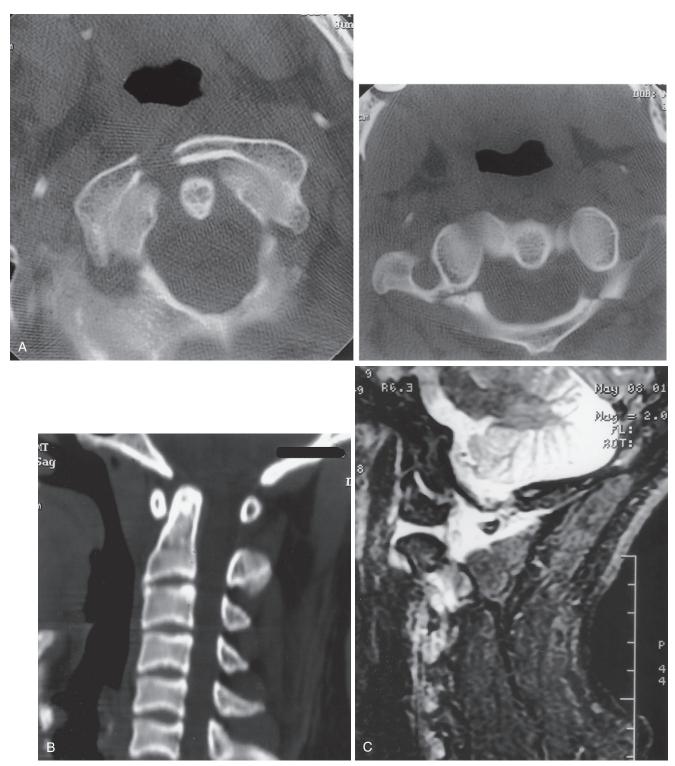
Fig. 16-2 *A,* This 14-year-old male patient sustained a complex fracture of the anterior arch of C1 and a bilateral lateral arch fracture of the atlas at the level of the vertebral artery grooves. *B,* Mid-sagittal computed tomographic reconstruction of the craniocervical junction shows the mild posterior displacement of the odontoid process in relation to the tip of the clivus. *C,* Parasagittal T2-weighted magnetic resonance image reveals significant ligamentous injury at the occipitoatlantoaxial level. *D,* Axial view T2-weighted magnetic resonance image obtained through the plane of the odontoid process at the level of the vertebral arteries. Note the preservation of the cruciate ligament behind the odontoid process.

Continued
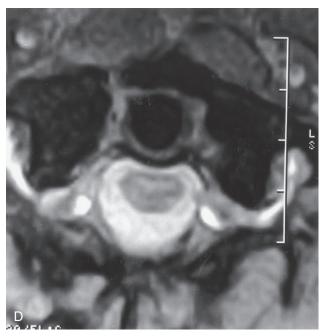
Fig. 16-2, cont'd *D*

fracture line usually extends to the superior articular facets of C2. Unlike adults for whom surgery might be indicated, type II fractures in the pediatric patient are easily treated with halo vests alone, achieving good rates of union.[33,50]

HANGMAN'S FRACTURE

Hangman's fracture is relatively uncommon in childhood. This injury typically is produced by hyperextension in which further movement of the C2 body is prevented by the inferior facets, resulting in a bilateral fracture in the weak pars interarticularis. After injury, the body of C1 and C2 often subluxes anteriorly. Plain radiographs and computed tomographic scans are useful for diagnosis, and treatment consists of halo immobilization after alignment is reestablished[33,61] (Fig. 16-4).

OCCIPITOATLANTAL DISLOCATIONS

Occipitoatlantal dislocations were once considered rare and nearly always fatal.[25,62,63] They continue to be associated with a high mortality rate.[41,44,64] Approximately 25% of fatal
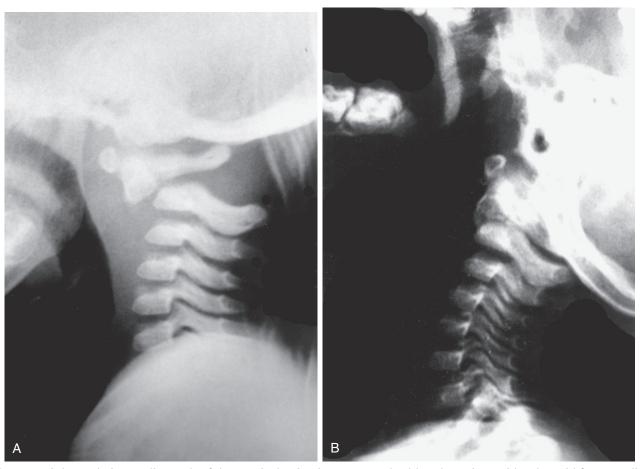
Fig. 16-3 *A,* Lateral view radiograph of the cervical spine in a 14-month-old male patient with odontoid fracture dislocation. Avulsion is present at the neurocentral synchondrosis. *B,* Radiograph of the same patient obtained 10 weeks after halo traction and immobilization. Healing with good alignment can be seen at the odontoid fracture site.
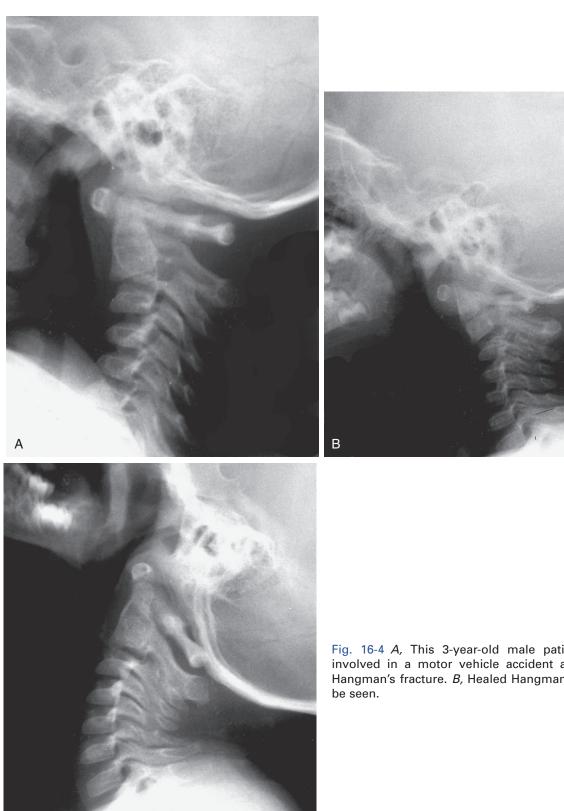
Fig. 16-4 *A,* This 3-year-old male patient had been involved in a motor vehicle accident and suffered a Hangman's fracture. *B,* Healed Hangman's fracture can be seen.

pediatric trauma can be attributed to traumatic occipitoatlantal dislocation.[65] Reports of occasional survivors have recommended dorsal occipital-cervical fusion to overcome the tremendous instability associated with these injuries.[63] However, MRI has enabled previously overlooked cases of occipitoatlantal dislocation with mild-to-moderate neurologic deficits to be identified, prompting a reevaluation of their management.

Several mechanisms have been implicated as the cause of occipitoatlantal dislocation; we think the most frequent mechanism is hyperflexion of the skull in relation to the upper cervical spine, with distraction. The resulting separation of the posterior elements of the atlas and axis is well visualized with the use of MRI. Ligamentous disruption of the anterior occipitoatlantal ligament, the tectorial membrane, the alar ligaments, and the posterior element of the occipitoatlantoaxial complex produces forward displacement of the cranium on the atlas. Although anterior occipitoatlantal injury is the most common, lateral occipitoatlantal dislocation and posterior cranial displacement have been reported.

Occipitoatlantal dislocations can be identified on plain radiographs by using various techniques.[62,63] One of the most widely accepted techniques is applying the Power ratio,[62] which is calculated by taking the distance between the basion to the posterior arch of C1 and dividing it by the distance between the opisthion and the anterior arch of C1. Under normal circumstances, this ratio averages 0.77, with ratios greater than 1 indicating pathologic abnormality. Because this method can be inaccurate in the case of posterior or longitudinal dislocations, we rely on MRI as the diagnostic gold standard; it can best identify ligamentous disruption, the often associated spinal cord and brainstem hemorrhagic contusion, and the possible presence of epidural hematoma external to the tectorial membrane.

Disruption of the tectorial membrane, however, can be manifested not only by occipitoatlantal dislocation but also by longitudinal C1-C2 distraction, because major segmental longitudinal stabilizers are lacking in the occipitoatlantoaxial complex.[7,66,67] As most of the extant radiologic criteria for diagnosing craniocervical instability focus on AOD dislocation, Sun et al.[7] recently proposed a technique using the C1-C2 to C2-C3 ratio to define the longitudinal C1-C2 interspinous relationship. In their series of 20 pediatric cases of occipitocervical trauma, a ratio of 2.5 or greater identified MRI-documented tectorial membrane abnormalities with a sensitivity rate of 86% and 100% specificity. A high index of suspicion must be held on evaluation of static plain films to help in the diagnosis of craniocervical instability, which must be confirmed by using MRI.

Most patients with occipitoatlantal dislocation have severe ligamentous injury and require occipital-cervical fusion,[63] especially those with longitudinal or posterior dislocations or with an initially unrecognized lesion that progresses to chronic instability. An acute anterior dislocation (without any longitudinal component) and associated mild deficit, however, has been treated in some pediatric patients with halo vests after reduction, with fusion reserved for those who fail external immobilization.

ATLANTOAXIAL INSTABILITY AND LUXATION

Instability of the atlantoaxial complex can present as anterior, posterior, or rotational luxations. Luxation refers to a complete and lasting disruption of the articular facets of the synovial joints. Examples of this are interlocking of the articular facets of the marked diastasis that occurs with hyperflexion fracture luxation.

Anterior or posterior subluxations often are associated with odontoid fractures; however, instability can develop from injury to the transverse and other associated ligaments.[33,40] Unlike the adult patient, in whom the predental space should not exceed 3 mm, pediatric patients might have predental spaces up to 5 mm under normal circumstances. If this dimension exceeds 5 mm, injury to the transverse ligament is suspected.[40,68] The integrity of the transverse ligament is important in treating anterior and posterior atlantoaxial instability, because patients with injury to the transverse ligament often require dorsal cervical fusion, whereas those with intact transverse ligament are treated successfully with halo immobilization.[33]

The rotatory atlantoaxial luxation injury is most commonly caused by neck trauma,[28,69,70] although it is not an uncommon finding in children with infections of the upper respiratory system or other inflammatory conditions or after surgical procedures on the head and neck or induction of general anesthesia.[28,40,71,72] Children with rotatory atlantoaxial luxation have been erroneously diagnosed as having brainstem vascular insult, cerebellar tumor, Chiari malformation, cervical migraine, syringohydromyelia, and ocular palsies.[40] All patients present with torticollis and diminished range of motion of the neck. An associated occipitoatlantal rotary luxation leads to a characteristic "cock-robin" appearance[56] (Fig. 16-5). Facial flattening is prominent and symptoms of neural compression occur when the atlas is separated from the odontoid process by more than 5 mm, allowing a rotation of the atlas and axis and thus compromise of the spinal canal.

In atlantoaxial rotatory luxation injuries, the skull and C1 are rotated on C2 with the dens serving as the axis of rotation. This region is difficult to visualize on plain radiographs, and CT of the craniovertebral junction with reconstructions often is required to clearly define rotation of the atlas on the axis.[33] Fielding et al.[29] classified atlantoaxial rotatory luxations depending on the integrity of the transverse ligament and other supporting ligamentous structures. With an intact transverse ligament, rotation will not exceed 35 degrees, and these injuries are easily realigned with traction and then halo immobilization. Often, these injuries resolve spontaneously if the initiating event was minor or atraumatic. However, when the transverse ligament is disrupted, rotation can

exceed 40 degrees, resulting in facet interlock.[33,40] The atlas arch is displaced forward, causing compromise of the spinal canal diameter, requiring open reduction with or without dorsal cervical fusion.[19,28,70,73–75] Here, closed reduction might be attempted first, but similarly, once it fails, open reduction should be performed in a timely manner to achieve the best improvement in head position and facial asymmetry[19,28,70,73–75] (Fig. 16-6).

SPINAL CORD INJURY WITHOUT RADIOGRAPHIC ABNORMALITY

Reports of spinal cord injury without visible fractures or dislocation were published in the literature as early as 1907.[76,77] Pang and Wilberger[10] were the first to coin the term *spinal cord injury without radiographic abnormality,* or SCIWORA. Reports on the incidence of SCIWORA vary widely, ranging from 5% to 70% of pediatric spinal injuries, much more common than in adults.[3,12,41,45–47] Often, the neurologic picture resembles a central cord syndrome, either presenting rapidly or in a delayed manner, and most cases involve the upper cervical spine.[10,20,41,46–48,78]
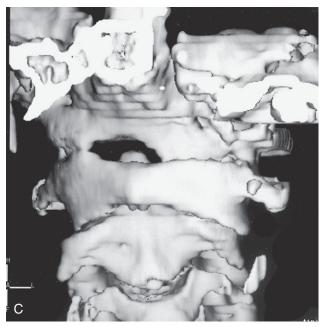
Older children with sports-related trauma[41] present with transient neurologic findings, some of which progress. After initial immobilization in a collar, it is imperative that flexion-extension films be obtained and MRI be performed, because surgical lesions such as a herniated disk or epidural hematoma can be excluded, just as fracture or instability is excluded via use of plain radiography and CT. MRI commonly reveals cord edema. It is not uncommon for children who are partially affected with neurologic dysfunction to have a recurrent episode a few weeks after the initial insult. For this reason, these children should be braced after the initial neurologic injury and kept so for 3 months to regain ligamentous stability.

Fig. 16-5 *A,* This 10-year-old female patient presented with a cocked head as with a "cock robin" appearance. Note the flattening of the left sternomastoid region and the tilting of the head. *B,* Frontal anteroposterior view radiograph of the head and cervical spine shows the cocked head position. *C,* Three-dimensional computed tomographic scan shows perching of the occipital condyle on the lateral mass of C1 on the right with a normal appearance and articulation of the occipital condyle-lateral atlantal mass on the left. *D,* Normal facial appearance and head and neck position after manual reduction of the occipitoatlantal dislocation.
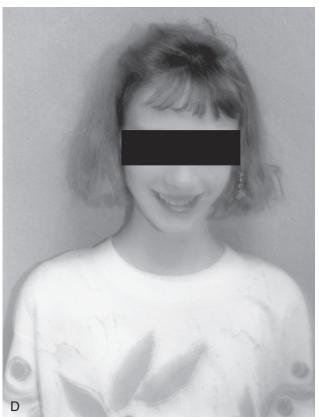
Fig. 16-5, cont'd *C* and *D*

OPERATIVE AND NONOPERATIVE MANAGEMENT OF TRAUMA

Reduction is best achieved with the head and neck placed in a hyperextended position over the edge of a mattress; if this does not succeed, halo ring immobilization should be implemented. Once adequate reduction of the spinal deformity has been achieved, rigid external immobilization can then be obtained by using a halo vest, which offers the advantage of prolonged immobilization and active early rehabilitation.

Skeletal traction in the toddler or young child can be applied safely provided attention is given to such factors as skull thickness, age, and anatomic and biomechanical properties of the pediatric spine. In older children, whose skull is of adequate thickness, traction can be applied by means of a standard halo ring or a crown halo. At our institution, MRI is performed acutely for all children with spinal cord injury to exclude a surgical lesion; therefore, MRI-compatible skeletal tongs or an MRI-compatible halo is preferred. After approximately 1 year of age, halo rings or crowns can be used with caution.

In children from 1 to 4 years of age, a halo ring can be used with eight pins for fixation to evenly distribute the forces at 2 pounds per square inch of pressure; however, caution is needed because skull perforation can occur.[11] In chil-

dren younger than 2 years, finger tightening is preferred. The amount of weight required to achieve reduction of cervical spine subluxation in children is considerably less than that needed in teenagers or adults; initially, it should not exceed 1 to 2 pounds in patients younger than 2 years. It is imperative that frequent films be obtained to ensure that overdistraction does not occur at the site of injury, which can result in a worsening of neurologic deficit or, in some instances, can promote the formation of an os odontoideum.[40,55,79] Once reduction is achieved, alignment must be maintained. Symptoms of local pain and headache might indicate penetration of the skull, leading to osteomyelitis, or even cerebritis.

Cervical spine fractures in older children are managed with reduction and immobilization. The presence of disk herniation, bone fragments, or hematoma impinging on the spinal cord requires decompression and fusion, either anteriorly or posteriorly.

When posterior fusion is necessary in the cervical spine, wire fixation alone does not suffice because the ultimate fusion construct should be osseous. Wire can cut through the thin, poorly ossified lamina or spinous process, which can lead to a position of hyperextension as the child grows or, even worse, subsequent disruption of the wire with impingement of the spinal cord.[6,40,47] The treatment of choice is wiring of bone to
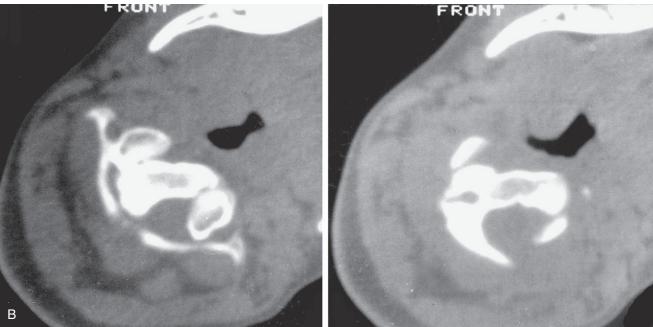
Fig. 16-6 *A,* Anteroposterior view radiograph of the skull and cervical spine in a 10-year-old male patient who experienced motor vehicle trauma. This child was unable to straighten his head and neck and had paresthesias in the arms with vertigo. The diagnosis was rotary dislocation of C1 on C2 with facet interlock of more than 45 degrees. *B,* Composite of axial view computed tomographic scans obtained through the plane of the atlantoaxial joint. Note the jumped facet with interlock on the right.

each individual lamina or facet, so that once incorporation of the bone has taken place, the bone will grow with the child. Our experience during the past 35 years has shown no decrease in the growth potential or exaggerated lordosis. From a neurosurgical perspective, the bone can be harvested from rib or posterior iliac crest or can be shaved from the occiput by using a micro-air impacter. We prefer to use rib or, if already exposed, occipital bone for posterior cervical fusions.

COMPLICATIONS AND AVOIDANCE

Aggressive surgical and medical management of spinal column and spinal cord injury combined with aggressive rehabilitation has led to an increased number of children surviving these devastating injuries. Many common problems observed in pediatric patients with paraplegia or quadriplegia parallel those observed in adults, including genitourinary tract infections, pneumonia, decubitus ulcers, and myositis ossificans.

Posttraumatic spinal deformity can occur at any age, although it seems to develop much more often in young children than in adolescents or adults. This complication, however, is more often attributable to injuries sustained to the subaxial cervical and thoracic spine. Unrecognized odontoid fractures might lead to delayed progressive cervical myelopathy if displaced ununited bony fractures are present; in these patients, transoral decompression and dorsal occipitalcervical fusion is tolerated better and is preferred over posterior fossa decompression and upper cervical laminectomy, which might increase the risk of spinal deformity.[80]

High cervical spinal injury in the child who has sustained a high-energy impact, especially a child who is younger than 8 years, often is associated with brainstem lesions or closed head injuries, which can present with initial apnea and/or cardiopulmonary arrest.[44,81,82]

Extracranial dissections have a low overall incidence of less than 0.4% in the entire trauma population,[83] but carotid artery dissection should be suspected in patients with severe craniocervical injury, especially those with delayed neurologic symptoms in the absence of visualized hematoma.[84–86] Although it can occur via several mechanisms, the most common is a direct blow to the head with hyperextensionrotation of the neck, which causes stretching of the internal carotid artery over the transverse processes of the first and second cervical vertebrae.[85,87] MRI, magnetic resonance angiography, or conventional angiography is necessary to diagnose these injuries for therapy to be initiated, which can vary from observation, anticoagulation, or antiplatelet therapy to endovascular or open surgery in certain select patients.

PROGNOSIS

Reports of outcomes after spinal cord injury in pediatric patients are mixed. Unfortunately, many patients with craniocervical injuries, especially those with occipitoatlantal dislocation, die at the scene or soon after presentation for medical care.[25,44,46,47,88] Although early diagnosis and prompt immobilization in some patients with AOD have made it potentially survivable,[64,65,89–91] when present with inaugural apnea or cardiopulmonary arrest at the scene, it indicates an associated high spinal cord or brainstem injury, and outcome is dismal.[81,82] The prognosis for recovery of neurologic function, though, varies substantially for other injuries, with some advocating that the "plasticity" of the pediatric central nervous system confers an improved diagnosis and others thinking that outcome is poor.[14,47,92–95] Perhaps the best predictor of good outcome is the initial neurologic status of the patient and the next best is expedient diagnosis and treatment.[96]

CONCLUSION

Because the spine is in a process of rapid development in children younger than 8 years, distinct differences exist in the pattern of spinal cord and spinal column injuries that occur during infancy and early childhood compared with those occurring in older children and adults. Young children especially tend to have injuries affecting the craniovertebral junction and upper cervical spine, more so than do adolescents and teenagers. Some features of the spine during early childhood offer protection, such as resistance to fractures; however, ligamentous laxity and incomplete development of bony structures lead to greater risk of subluxation.

Although many pediatric patients succumb to their injuries at the scene, improvements in emergency medical services and treatment, combined with the recognition of important differences between pediatric and adult spinal columns and cord injuries, has improved survival. Most injuries can be managed nonoperatively with halo immobilization or other orthoses. Care of these children does not end with their discharge from the hospital. Aggressive follow-up is required for early diagnosis of any possible complicating factors, such as progressive spinal deformity or progressive neurologic deficit.

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Atlas Fractures

INTRODUCTION

Fractures of the atlas were originally described in 1822 by Sir Ashley Cooper and current studies indicate that approximately 10% of cervical fractures involve the first vertebra.[1,2] A century following the initial description, Dr. Jefferson[3] published his classic paper "Fracture of the Atlas Vertebra" in which he presented his experience with four cases and reviewed the available literature. In his article Dr. Jefferson correctly reported that fractures of the atlas are caused either by axial loading or hyperextension and he characterized the "Jefferson fracture" which involves a burst fracture of the atlas ring. Following the 1920 publication of Dr. Jefferson's article, a number of clinical, radiographic, and biomechanical studies have been presented evaluating fractures of the atlas. Although these articles have significantly expanded the available knowledge regarding atlas fractures, current treatment remains based on Class III evidence consisting primarily of case series and reports. This chapter seeks to summarize our existing understanding of C1 fractures, but it must be made clear from the outset that evidence-based studies have not yet been performed that provide Class I or Class II data addressing the management of patients with fractures of the atlas vertebra.[4]

ANATOMY

To understand fractures of the atlas one must appreciate the anatomy of this unique vertebra and its surrounding structures. Upwards of 50% of cervical motion occurs through the cervicocranial complex, which consists of the bones formed by the occiput, atlas, and axis.[5] Being the first of the seven cervical vertebrae, the atlas forms the central portion of the cervicocranium and is responsible for transmitting loads from the skull to the lower cervical spine. One of the unique features of the atlas is that it has no true vertebral body. Its vertebral body has been incorporated into the axis in the form of the dens. Steele described the anatomic relationship at the level of the atlas in which the spinal canal consists of one-third odontoid process, one-third spinal cord, and one-third space (Fig. 17-1). This has come to be known as the "Steel's rule of thirds."[6] As discussed later, the significant amount of space present in the spinal canal at the level of the atlas has a significant impact on the clinical presentation and treatment of patients with fractures of the C1 vertebra.

In place of a vertebral body, the atlas has two large lateral masses. The two lateral masses form the articulations with the occipital condyles above and the superior articular facets of the axis below. These articulations are located anterolaterally in reference to the spinal canal, whereas the articulations of C3 and below are located posterolaterally. All of the articular surfaces of the atlas are concave. The articular facets for the occipital condyles face upward and inward while the articular facets for the axis face downward and inward and are relatively flatter. Anteriorly the lateral masses of the atlas coalesce to form a very thick anterior arch. The anterior arch contains a tubercle for the insertion of the anterior longitudinal ligament and longus colli muscles.

Posterior to the lateral masses is the relatively thin posterior arch. The atlas has no true spinous process, only a small tubercle that serves as the attachment site for the ligamentum nuchae. The thinnest portion of the posterior arch is located immediately posterior to the lateral masses. This is the region where the vertebral artery crosses over the posterior arch after having exited from the transverse foramen of the atlas. In most cases there is an identifiable groove in this location (Fig. 17-2). Care must be taken when surgically exposing the posterior ring of C1 to avoid damaging the vertebral artery, which is typically located at least 1.5 cm lateral to the midline in adults and 1 cm lateral to the midline in children. The lateral most portion of the atlas is comprised of the transverse foramen containing the vertebral artery and the transverse process, which serves as a site for muscular attachments.

The cervicocranium relies on both its osseous structures and ligamentous structures for stability. There are no intervertebral disks present between the occiput, atlas, and axis. Because of this fact, the ligamentous anatomy in this region is of paramount importance for stability. The ligamentous anatomy can be divided into an external and an internal component. The external cranio-cervical ligaments are composed of the ligamentum nuchae, anterior and posterior
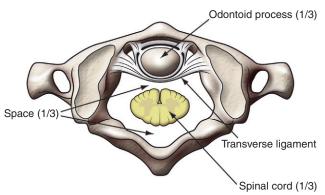
Fig. 17-1 Superior view of the atlas. Note the relationship of the odontoid process, transverse ligament and spinal cord. Steele "rule of thirds" describes the fact that the spinal canal at the level of the atlas is occupied by one-third odontoid process, one-third spinal cord, and one-third space.
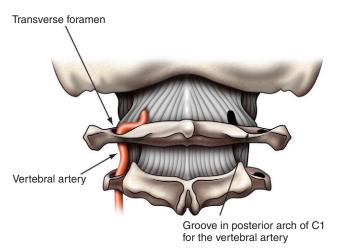

Fig. 17-2 Posterior view of the craniocervical junction. Note the vertebral artery exiting the transverse foramen and crossing the posterior arch of the atlas.

atlanto-occipital ligaments, and the anterior and posterior atlanto-axial ligaments.[7] The ligamentum nuchae is a mostly collagenous tissue attaching to the external occipital protuberance, posterior tubercle of the atlas, and the remaining cervical spinous processes. The anterior atlanto-occipital ligament courses between the cephalad portion of the atlas and a tubercle at the base of the skull just anterior to the foramen magnum. This may represent a continuation of the anterior longitudinal ligament, which is not as apparent in this region. Posteriorly, the atlanto-occipital ligament courses between the posterior arch of the atlas and the posterior portion of the foramen magnum. Similar fibroelastic bands are present between the atlas and axis. All of these external cranio-cervical ligaments contribute very little to the structural stability of this region when compared with the internal ligaments.[8]

The internal ligaments of the cervicocranium are located within the spinal canal and provide the majority of the ligamentous stability. These ligaments form three distinct layers anterior to the dura. From posterior to anterior they include the tectorial membrane, the transverse atlantal ligament and the odontoid ligaments. The tectorial membrane is the continuation of the posterior longitudinal membrane and connects the posterior body of the axis to the anterior foramen magnum. Anterior to the tectorial membrane is the transverse atlantal ligament. The transverse ligament is the most important ligamentous structure in the cervicocranium. It extends transversely from one condyle of the atlas to the other and also has ascending and descending vertical bands that attach to the foramen magnum and the body of the axis respectively (Fig. 17- 3). This ligament is mainly responsible for preventing anterior dislocation of C1 on C2 and preventing posterior migration of the odontoid process into the spinal cord.[9] Anterior to the transverse ligament are the alar and apical ligaments. The alar ligaments are a pair of structures that connect the superolateral odontoid process to the anterior-medial surface of the occipital condyles. The alar ligaments limit axial rotation to the opposite side and provide stability to the atlas although they do not attach directly to it.[10] The apical ligament connects the tip of the dens to the anterior portion of the foramen magnum and is relatively small in comparison to the alar and transverse ligaments.

In addition to the ligamentous stabilizers, muscular attachments play a role in providing stability to the craniocervical region. The superior portion of the longus colli muscle attaches to the anteroinferior aspect of C1. More posteriorly, the rectus capitis lateralis and medialis run from the transverse processes of C1 to the lateral and anterior portion of the foramen magnum, respectively. The rectus capitis posterior muscles run from the posterior arch of
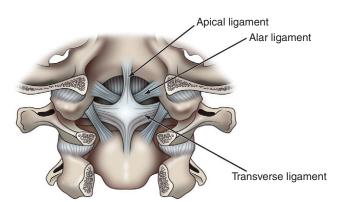

Fig. 17-3 Ligamentous anatomy of the craniocervical junction as viewed when looking anteriorly from within the spinal canal. In this diagram, the odontoid process is obscured by the transverse ligament, which is seen spanning the canal from one condyle of the atlas to the other. The apical and alar ligaments extend downward for the occiput to insert onto the odontoid process.

C1 to the occiput. Laterally, the superior oblique muscles connect the lateral aspect of the C1 ring to the occiput. More superficially, the cervical portion of the trapezius muscle also attaches to the posterior arch of C1 providing additional stability.

MECHANISM OF INJURY

Because it has been shown that specific mechanisms of injury are associated with particular atlas fracture patterns, knowledge of the mechanism will help in evaluating patients and in recognizing potential instability and associated injuries. Most Jefferson fractures are thought to occur through an axial load that is often transmitted via a direct blow to the head. Panjabi et al. [11] performed axial loading tests on cadaver cervical specimens and were able to create specific atlas fractures. They showed that a pure axial load produced an atlas fracture at 3050 N while the cervical spine was in a neutral position, and that only 2000 N was necessary to produce an atlas fracture with the spine extended. Finite elemental testing has further demonstrated that significant stresses and strains occur in the anterior and posterior arches during compressive loading of the atlas.[12,13] With axial loading, the occipital condyles are driven distally into the atlas in a wedge-like fashion fracturing the ring of C1 and displacing of the lateral masses outward.

Spence et al.[14] demonstrated in anatomic specimens that if the combined sum of left and right C1 lateral mass displacement (LMD) is greater than 6.9 mm, then the transverse ligament is most likely disrupted. The instability that results from rupture of the transverse ligament occurring in association with atlas burst fractures is not the same as the instability that occurs when a severe flexion injury leads to failure of the transverse ligament.[2] C1 burst fractures result in tensile failure of the transverse ligament as the occipital condyles splay the atlas fracture fragments apart laterally. With this mechanism of injury, the secondary restraints to excessive flexion including the alar ligaments, the apical ligament, and the C1-C2 capsular attachments are not disrupted. In contrast, although there are no fractures, severe flexion injuries resulting in rupture of the transverse ligament also typically disrupt the alar and apical ligaments and the capsules of the C1-C2 articulation. The integrity of the secondary stabilizers in atlas fractures significantly affects atlanto-axial stability and affects the management of the associated transverse ligament injuries.

Fractures of the posterior arch of the atlas are caused by a hyperextension mechanism in which the posterior arch of C1 is caught between the occiput and the posterior arch of C2 with the anterior aspect of C1 becoming fixed.[15] A subsequent bending moment is created when the occiput impinges against the posterior arch of C1. Fracture usually occurs in the thinnest portion of the ring where the vertebral artery crosses the arch of C1. This hyperextension mechanism helps explain some of the associated injuries that are often seen with posterior arch fractures, including traumatic spondylolisthesis of the axis and anterior teardrop fracture of the C2 vertebra.[5]

The transverse anterior arch fracture of C1 is also caused by a hyperextension mechanism. In this injury pattern, the anterior neck musculature contracts in response to sudden hyperextension causing the longus colli muscle to avulse the anterior inferior portion of the atlas where the superior oblique portion of the muscle attaches to the anterior tubercle. This pattern is relatively stable because it represents an avulsion and not a failure through the entire anterior arch of the atlas or the dens. The initial lateral radiograph must be scrutinized closely as this fracture can potentially be mistaken for a more serious injury in which there is disruption of the C1-C2 complex.

C1 lateral mass fractures are an asymmetric injury pattern that usually results from a combination of axial loading with lateral bending or rotation. This mechanism produces ipsilateral fracture lines anterior and posterior to the lateral mass (Fig. 17-4). Occasionally, a third fracture line may be present in the posterior arch indicating that there may have also been a slight hyperextension moment associated with the injury. Significant displacement of the lateral mass can occur with unilateral disruption of the capsular structures. Facet fractures of the lower cervical spine occur by a similar mechanism and are occasionally found in association with lateral mass fractures. Forced lateral bending of the spine can also produce isolated transverse process fractures of the atlas although some transverse process fractures represent avulsions caused by the muscles inserting onto the lateral aspect of the C1 vertebra.
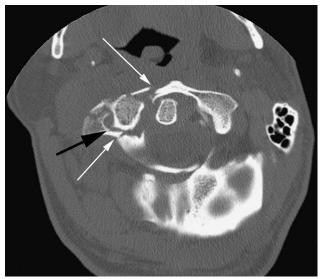

Fig. 17-4 Axial CT image of a lateral mass fracture. *White arrows* indicate fracture lines anterior and posterior to the lateral mass. The *black arrow* indicates fracture lines extending into the transverse foramen. Minimal lateral displacement of lateral mass has occurred.

CLASSIFICATION

Jefferson initially described two types of fractures of the atlas including a first group of isolated posterior arch fractures and a second group of burst fractures involving both the anterior and posterior arches.[3] Jefferson's name is associated with the four-part burst fracture of the atlas in which there are two fracture lines in each of the anterior and posterior arches. The classification of atlas fractures has changed over time and our understanding advanced significantly with the widespread introduction of computed tomography (CT) scanning. Atlas fractures are now generally classified using a combination of the location of the injury, mechanism of injury, and integrity of the transverse ligament. Clinical and anatomic studies have lead to the current system in which there are five generally recognized types of atlas fractures.[16–18]

The most common type of atlas fracture is the two-part break through the posterior arch. As noted earlier, the vertebral artery courses over the posterior ring of C1 at the junction of the lateral mass and the posterior arch leaving this area of bone with a thinner diameter. When a hyperextension injury produces a bending moment in the atlas ring, most fractures will occur through this relatively weaker area of the bone.[2,18,19] Care must be taken to distinguish posterior arch fractures from a congenital failure of development or incomplete ossification of the posterior ring of C1.

The second type of atlas fracture is the classic Jefferson four-part burst fracture (Fig. 17-5). This injury usually results in splaying of the atlas in a radial fashion and increases the diameter of the spinal canal at this level. There are variants of the Jefferson fracture, which can have two or three parts.[16,20] The two-part fracture has midline breaks through both the anterior and posterior arches. The three-part variant
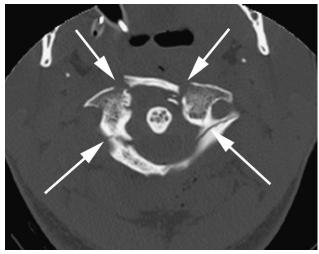

Fig. 17-5 Axial CT image of a Jefferson fracture. *White arrows* indicate the four fracture lines through the anterior and posterior rings. Some posterior migration of the odontoid process is noted.

will usually have two fractures in the posterior arch and one in the anterior arch. The Jefferson fracture can be further classified as stable or unstable based on the integrity of the transverse ligament, as discussed later in this chapter.[14]

The third type of atlas fracture involves ipsilateral breaks in the C1 ring just anterior and posterior to one of the lateral masses. When compared these lateral mass fractures, Jefferson fractures tend to have symmetrical lateral displacement, whereas lateral mass fractures will have asymmetrical displacement. Lateral mass fractures can potentially be unstable if they are markedly displaced or comminuted. A fourth type of atlas fracture involves a break in the transverse process portion of the lateral mass only. These are stable injuries but can be associated with damage to the vertebral artery.[21]

The final type of C1 fracture occurs in the transverse plane through the anterior arch of the atlas and represents an avulsion by the longus colli muscle. This injury is a stable injury that must be distinguished from the more serious bilateral anterior arch fracture associated with posterior atlanto-axial dislocation. Those potentially unstable injuries are termed plough fractures in which the dens is forced anteriorly through the arch of the atlas.[22] CT scanning with axial cuts and sagittal reformations will allow these fracture patterns to be distinguished.

CLINICAL PRESENTATION

Atlas fractures commonly occur in young adults and are often the result of significant trauma such as that caused by motor vehicle accidents, diving injuries, or direct blows to the head. Patients that are awake usually present with a history of trauma, suboccipital headache, and neck pain or stiffness. Neurologic symptoms are rare in isolated atlas fractures because the injury tends to expand the bony ring of C1 outward and because the spinal canal is large relative to the spinal cord at the level of C1.[23,24] Associated head injuries can make patients with C1 fractures difficult to examine. It is also important to note that fractures of the atlas are often associated with other cervical spine injuries with the reported prevalence of concomitant cervical fractures ranges from 30% to 70%.[7,25] The three most significant associated injuries include fractures of the dens, traumatic spondylolisthesis of the axis, and burst fractures of the lower cervical spine.[26,27] Patients may have neurologic symptoms related to these other fractures and not necessarily from neurologic injury secondary to the atlas fracture itself.

When a patient with a possible atlas fracture presents to the emergency room, a complete physical examination of the patient should be carried out with the patient immobilized in a cervical collar and care taken to maintain neutral alignment of the spine. A thorough neurologic examination must be performed including testing of the cranial nerves because there have been documented cases of cranial nerve injuries associated with atlas fractures.[28,29] Injury to the

vertebral artery can occur in association with fractures of the atlas, so signs of a stroke should also be sought.[30] Because the physical findings of atlas fractures can be subtle and neurologic deficits in isolated C1 fractures are rare, these injuries can initially be missed if one is not cautious.[5,31]

DIAGNOSTIC STUDIES

Standard radiographs of the cervical spine include antero-posterior (AP), lateral, and open-mouth odontoid images. The screening lateral C-spine image may identify a fracture through the posterior arch of C1 or an avulsion fracture of the anterior arch. An increase in the prevertebral soft tissue shadow is a more subtle sign of possible significant injury to the atlas. In adults, the prevertebral shadow anterior to the C2 vertebra typically should not exceed 5 mm in the neutral position.[32] This sign has been shown to be somewhat unreliable in children and in patients who are being evaluated within the first hour of the injury. Complex fractures of the atlas may not be entirely recognized on the lateral view. An open-mouth or odontoid view should also be obtained to identify displacement of the lateral masses of C1 on C2. As noted earlier, Spence has shown that if there is a combined displacement of more than 6.9 mm, then the transverse ligament is likely to have been ruptured.[14] More recently, Heller et al.[33] have demonstrated an average magnification factor of 18% on standard open-mouth/odontoid view x-rays. Applying this factor to the rule of Spence suggests that the sum of the LMD of C1 over C2 as measured on open-mouth odontoid radiographs should be raised to greater than 8.1 mm to indicate a likely disruption of the transverse ligament (Fig. 17-6).

Although most texts and classic articles focus on plain radiographs, recent studies in trauma patients have found that CT scanning of the cervical spine is quicker and more accurate than standard x-rays.[34] CT scanning with sagittal and coronal image reconstruction has become the standard method of evaluating suspected injuries to the upper cervical spine.[35] A high-resolution CT scan permits precise evaluation of an atlas fracture's location, degree of comminution, and displacement. Given the high incidence of associated cervical fractures, all patients who sustain an atlas fracture should under-go CT scanning of their entire cervical spine.

Magnetic resonance imaging (MRI) has been increasingly used to evaluate injuries to the upper cervical spine.[36] The primary value of an MRI in patients with atlas fractures is to evaluate for the presence of an injury to the transverse ligament. In a study looking at isolated transverse ligament ruptures, Dickman et al.[37] identified two types of ligament injuries based on MRI findings. Type I injuries involve an intrasubstance ligament tear with no associated fracture. Type II injuries include an avulsion fracture at the insertion site of the transverse ligament. The authors concluded that type I injuries were inherently unstable and required internal fixation and that type II injuries had a higher chance of healing and should be treated with rigid external immobilization. Because Dickman's review did not look at patients with atlas fractures, it is unknown whether their conclusions can be extrapolated to the care of these patients. Given the lack of studies on the topic, the current role of MRI in the evaluation of patients with atlas fractures remains poorly defined.
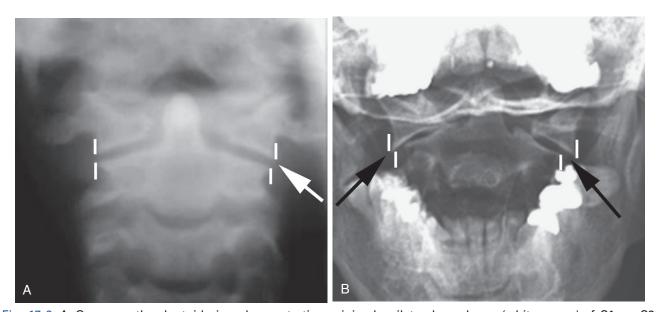

Fig. 17-6 *A,* Open-mouth odontoid view demonstrating minimal unilateral overhang *(white arrow)* of C1 on C2. *B,* Open mouth odontoid view in another patient demonstrating significant bilateral overhang *(black arrows)* of C1 on C2. Injury to the transverse ligament is likely when the combined overhang anatomically exceeds 6.9 mm (8.1 mm when measured on open-mouth odontoid views to correct for the usual amount of x-ray magnification).

TREATMENT

Two issues have complicated our ability to determine the proper treatment for patients with fractures of the atlas. The first problem involves the high number of associated cervical spine injuries that occurs in combination with fractures of the atlas. The patient's treatment often will be dictated by the associated injury and not the atlas fracture itself. The second concern involves the lack of prospective randomized trials evaluating the various treatment modalities available for fractures of the atlas. As noted at the outset of this chapter, all of the available clinical treatment guidelines have been drawn from case series and retrospective reviews. The primary goals of treatment that must be considered are ensuring that fracture healing occurs while maintaining patient comfort, preventing further harm, and adequately treating any associated injuries. Because the majority of atlas fractures are stable injuries and have no associated neurologic compromise, these goals can usually be achieved via external immobilization in either a cervical collar or a halo vest with formal operative intervention only rarely necessary.

The most common type of atlas fractures are isolated breaks in the posterior arch. These are inherently stable injuries because there are no major ligamentous disruptions. Posterior arch fractures are usually minimally displaced and tend to have a very high rate of union. These fractures typically heal in 2 to 3 months with callus often visible on plain radiographs at that point. Minimal immobilization has been found to be necessary with most authors recommending a rigid cervical collar for 12 weeks.[38]

The treatment of posterior arch fractures becomes more complicated in the presence of associated cervical spine injuries. The two most commonly associated injuries are fractures of the dens and traumatic spondylolisthesis of the axis. A type II posteriorly displaced odontoid fracture can be particularly challenging because these injuries have a higher rate of nonunion and late displacement than the other types of dens fractures. One option for treatment is an initial closed reduction of the dens fracture followed by rigid immobilization in a halo vest for 2 to 3 months. A second option that is becoming more widespread is screw osteosynthesis of the dens fracture followed by immobilization in a rigid cervical collar for three months.[27] Screw fixation of the odontoid raises the union rate and obviates the need for use of a halo vest while preserving C1-C2 motion (Fig.17-7). The third option for management of a combined type II odontoid fracture and a posterior arch fracture of the atlas is immediate C1-C2 posterior arthrodesis. Because the posterior arch of C1 is disrupted in this scenario, the C1-C2 fusion construct requires either transarticular screw fixation or the use of a C1 lateral mass and C2 pedicle screw construct. Posterior C1-C2 arthrodesis in the presence of a C1 posterior arch fracture is technically demanding and will result in loss of the significant cervical rotation associated with a functional C1-C2 segment. Occipitocervical fusion for the combined C1 posterior arch
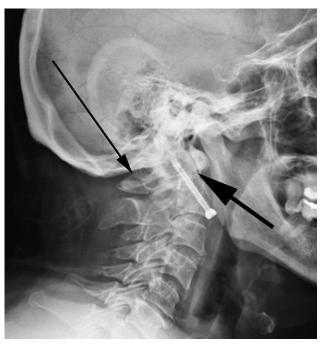

Fig. 17-7 Combined posterior arch atlas fracture *(thin arrow)* with a type II odontoid fracture *(side arrow)* treated with anterior odontoid screw fixation.

and type II odontoid fracture is a final option if odontoid screw fixation and C1-C2 fusion are not technically feasible, but this alternative should rarely prove necessary with currently available instrumentation constructs. Another injury often occurring in combination with a posterior arch of the atlas fracture is a type I traumatic spondylolisthesis of the axis. Each of these fractures is separately stable and typically only requires immobilization in a cervical collar. When they occur concurrently, the overall stability remains unchanged and the treatment remains a rigid cervical collar for 3 months.

Management of avulsion fractures of the anterior ring of the atlas or transverse processes can usually proceed in a symptomatic manner with immobilization in a cervical orthosis. These injuries are stable and should not cause any long-term morbidity. Most lateral mass fractures of the atlas are minimally displaced, and for this reason they can also be effectively treated in a cervical orthosis for 8 to 12 weeks. Follow-up radiographs should be obtained to look for any further displacement or C1-C2 instability. It has been shown that significantly displaced or comminuted lateral mass fractures can be treated with either a halo vest or cervical collar.[4] The most severe lateral mass fractures with wide displacement should be treated similarly to Jefferson fractures as described later.

Treatment of the classic Jefferson burst fracture is related to the displacement of the fracture and associated transverse ligament injury. The Jefferson fracture can be divided into three main types: non-displaced, minimally displaced, and

significantly displaced. The minimally displaced fracture has less than 7 mm of combined LMD and the significantly displaced fracture has greater than 7 mm of displacement. If no displacement is noted on the open-mouth view or coronal CT scan, then treatment is either in a cervical orthosis or halo vest for 12 weeks. Studies have shown high union rates associated with this type of fracture. Minimally displaced fractures can be treated in a similar manner. In both of these types of injuries the intact transverse ligament will maintain the relationship of C1 and C2.[9] There is some concern that the displacement and incongruity of the atlanto-occipital and atlanto-axial joints could lead to future arthrosis. It has been hypothesized that the shallow shape of these joints may allow for a significant degree of incongruity to be well tolerated. To date, there have been no studies correlating the amount of C1-C2 displacement and development of arthrosis.

Jefferson fractures with greater than 7 mm of combined LMD have a presumed transverse ligament injury. As noted earlier, the secondary stabilizers of the atlanto-axial joint, including the apical and alar ligaments and the facet capsules, are typically intact. There are various methods described to treat displaced burst fractures of the atlas with presumed rupture of the transverse ligament. The first method involves halo vest placement for 12 weeks followed by flexion-extension views of the cervical spine (Fig. 17-8). If significant C1-C2 instability persists, then a C1-C2 posterior arthrodesis is recommended. A second option requires

prolonged hospitalization and entails performing a closed reduction of the atlas fracture using cranial traction. The reduction is confirmed using open-mouth odontoid x-rays. After 4 to 6 weeks of traction the fracture callus typically matures to the point that the reduction can be maintained without traction. A cervical collar or halo is then applied to complete a total treatment course of 12 weeks. Flexion-extension views of the cervical spine are then obtained to assess for any persistent C1-C2 instability.

Other treatment options for the displaced Jefferson fracture include performing an immediate C1-C2 posterior arthrodesis. This option reduces the risk of a patient going through prolonged immobilization for the initial fracture management followed by a second period of immobilization should additional treatment be necessary for any persistent C1-C2 instability. Primary C1-C2 arthrodesis also improves the congruity of the atlanto-occipital joint, which may help prevent future degenerative disease. Performing a primary occiput to C2 arthrodesis has also been described as a treatment of the widely displaced Jefferson fracture, but efforts should be taken to avoid this fairly morbid fusion of the entire craniocervical region if at all possible. Even in difficult fractures, patients can usually be immobilized in a halo until fracture healing occurs, followed by a delayed C1-C2 fusion to address and persistent instability. This option preserves the motion associated with the important occipito-cervical articulation.
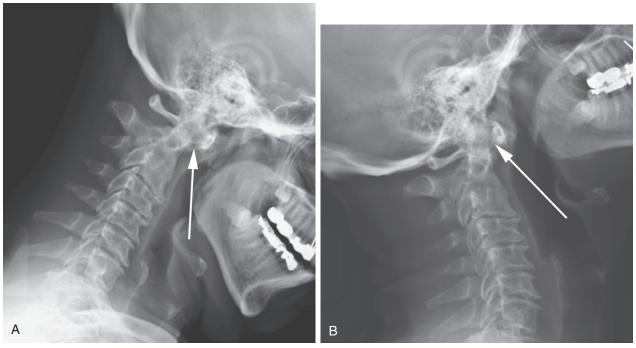

Fig. 17-8 Lateral flexion *(A)* and extension *(B)* x-rays obtained 3 months following closed management of a displaced Jefferson fracture. Some increase in the atlanto-dental interval *(white arrow)* is noted with flexion that reduces on extension. This mild degree of instability is consistent with a transverse ligament injury that has occurred while the secondary stabilizers, including the apical and alar ligaments as well as the C1-C2 facet capsules have remained intact.

Hadley et al. evaluated 32 patients with isolated atlas fractures.[24] Five of these patients had unstable Jefferson fractures with a combined LMD of greater than 7 mm. These five patients were treated in a halo vest for a period of 12 to 16 weeks. There were 25 patients with either nondisplaced or minimally displaced fractures who were treated with either a cervical collar or a suboccipital-mandibular immobilizer (SOMI) for 8 to 12 weeks. No patient demonstrated signs of instability or nonunion and none required subsequent surgical fixation. The authors concluded that atlas fractures with a LMD of more than 7 mm required more rigid immobilization in a halo vest and those fractures with an LMD of less than 7 mm required only a cervical orthosis.

Fowler et al.[23] evaluated 48 patients with atlas fractures of which 30 were classic Jefferson type of fractures. Their algorithm called for closed reduction maintained by cranial traction for 4 to 6 weeks for all patients with greater than 7 mm of LMD. Following traction the patients were placed into a halo for a total treatment time of 12 weeks. All of these patients had bony union and none required further surgery. The authors devised this algorithm based on the findings of a study by Zimmerman in which it was demonstrated that immediate immobilization in a halo did not achieve or maintain reduction of fractures with significant displacement of the lateral masses.[39] Zimmerman's paper established that because the primary mechanism to produce this fracture is axial loading, constant axial traction is necessary to maintain the reduction.

Other authors have noted that treating displaced atlas fractures with immediate C1-C2 arthrodesis offers the advantage of avoiding prolonged traction or treatment in a halo. One technique requires that closed reduction be obtained in traction followed by placement of C1-C2 transarticular screws. McGuire and Harkey[40,41] described two cases of unstable Jefferson fractures with LMD of greater than 7 mm treated with this construct. Both patients in their series achieved good results using only a cervical collar for postoperative immobilization. The authors concluded that C1-C2 arthrodesis eliminated the need for halo immobilization and is an attractive alternative in older patients or those at high risk with prolonged traction and bed rest. Other small series have validated the results of primary C1-C2 arthrodesis followed by use of a cervical orthosis for up to 12 weeks.[18,42,43]

A newer treatment for widely displaced Jefferson fractures has recently been described by Ruf et al.[44] The authors identified six patients with unstable Jefferson fractures treated with transoral reduction and primary C1 osteosynthesis. The patients had an average LMD of 13.5 mm preoperatively that was reduced to 4.3 mm postoperatively. The mobility of the C1-C2 joints and the congruency of all of the articular surfaces involved were evaluated with MRI. The authors concluded that this new technique allowed maintenance of the C1-C2 joint's rotatory mobility and restored the congruency of the atlanto-occipital and atlanto-axial joints. No patient in their series had any postoperative C1-C2 instability.

When looking at overall outcomes of patients with fractures of the atlas, short-term follow-up indicates a good outcome, but there is a scarcity of studies with long-term functional follow-up on these patients. As noted earlier, there is an unresolved question as to whether the persistent incongruity of the atlanto-occipital and atlanto-axial articulations that exists in unreduced atlas fractures has a negative impact on long-term outcome. Because it has been suggested that a number of patients do go on to develop painful osteoarthritis following displaced atlas fractures, controlled studies to assess this issue must be performed.[45] If persistent joint incongruity is found to significantly increase the risk of long-term problems, then treatment algorithms should be shifted more toward the operative options involving traction, primary C1-C2 arthrodesis, or direct anterior C1 osteosynthesis and away from nonoperative management with external immobilization.

CONCLUSION

The majority of fractures involving the atlas are stable and are not associated with any neurologic compromise. Because there are few specific physical examination findings in patients with atlas fractures, a high index of suspicion based on the mechanism of injury must be maintained. Radiographs and CT imaging with reconstructions will confirm the diagnosis and allow the fracture to be properly classified. Proper imaging will also detect any of the other injuries to the cervical spine that are often associated with atlas fractures.

Initial management of atlas fractures involves cervical immobilization in a rigid orthosis. Isolated posterior arch fractures, transverse process fractures, anterior tubercle fractures and nondisplaced lateral mass fractures are all stable injuries that can be managed nonoperatively. These fractures typically heal without incident and have not been shown to cause serious complications. A more serious injury is the three- or four- part burst fracture of the atlas caused by axial loading that typically leads to splaying of the C1 ring on C2. The most important factor associated with the treatment of C1 burst type fractures is the integrity of the transverse ligament. If the combined LMD of C1 on C2 is less than 6.9 mm, then the transverse ligament typically is intact and the injury can be treated nonoperatively in a cervical orthosis. If the LMD is greater than 6.9 mm, then the treatment is either a halo vest, reduction with traction, or C1-C2 arthrodesis.

Because even widely displaced atlas fractures can usually be managed with a halo vest, large series of operatively treated patients do not exist. In patients managed nonoperatively, care must be taken following immobilization to obtain flexion-extension radiographs to determine if there is any persistent C1-C2 instability. If instability is noted at that point, a C1-C2 arthrodesis can be performed using any one of the described techniques. When immediate C1-C2

arthrodesis is chosen for treatment of displaced atlas fractures, either C1-C2 transarticular screws or C1 lateral mass and C2 pedicle screws must be used because the presence of the fracture precludes wiring techniques to the posterior arch of the atlas. A more recently described technique involving a direct transoral approach and C1 osteosynthesis also exists, but wide experience with this treatment option does not exist. As in many areas of spine surgery, atlas fracture management continues to evolve and there remains a significant need for comparative studies evaluating the various treatment options.

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C H A P T E R 18

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Transverse Atlantal Ligament Injuries

INTRODUCTION

The upper cervical spine consists of the atlas, the axis, and interacting ligamentous structures. These two uniquely shaped vertebrae (C1 and C2) maintain their anatomic relationship through a very complex multilayered ligamentous system extending between the skull base and the third cervical vertebra. Injury to the osseoligamentous components can compromise the structural integrity of the entire craniocervical region. Because of the presence of vital neurovascular structures located in this region of the spine, the integrity of this region is of vital importance.[1,2]
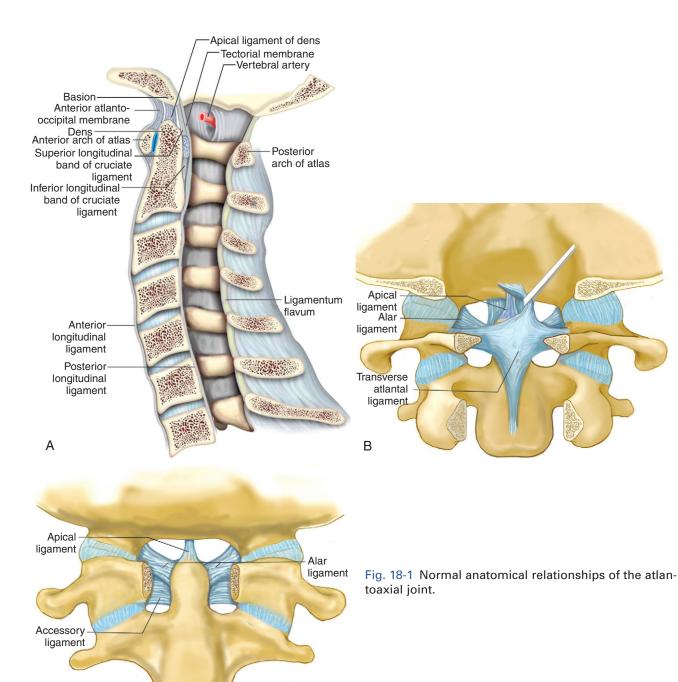
Traumatic ligamentous injuries of the atlanto-occipital joint and transverse atlantal ligament (TAL) are relatively uncommon. Acute traumatic rupture of the transverse ligament can often be fatal and is often associated with head injuries. Rupture of transverse ligament can occur in isolation or with atlantoaxial subluxation or an atlas fracture. The transverse ligament can fail in mid-substance or avulsion of bone can occur.[3,4] TAL injuries associated with avulsion fracture of the lateral mass tubercle was first reported by Barker et al.[5] in four adult patients. These injuries occur in less than 10% of pediatric cervical spine injuries.[2,6] The complex regional anatomy and the overlying structures of the upper cervical spine make radiographic diagnosis of such injuries challenging. With better understanding of the mechanisms of injury, a heightened clinical awareness, and better diagnostic technology, TAL injuries are beginning to be recognized more often. Recommendations for treatment must reflect a proper understanding of the clinical presentation, radiographic assessment, and regional anatomy and mechanisms of injury to minimize the morbidity that accompanies unrecognized injuries of this region. This chapter is intended to synthesize current information about TAL injuries and their management.

PRESENTATION AND CLINICAL FEATURES

Isolated injury of the transverse ligament may occur as a result of acute traumatic rupture or simply by attrition in the presence of certain inflammatory conditions (e.g., rheumatoid arthritis). It is also often associated with burst fracture of the atlas (Jefferson fracture) and odontoid process fractures.[3,7–11] TAL rupture occurs in 10% of patients with odontoid fractures.[3,9] Because of the anatomical proportions and relations present in the child, the upper cervical spine is more prone to insults compared with other levels of the spine.[6]

Traumatic rupture of the TAL is probably by shear force applied to the posterior aspect of the head. Clinical reports suggest that force flexion may be involved. Often this results in transverse ligament rupture and some degree of injury to the alar and apical ligaments. These ligaments are responsible for atlantoaxial stability (Fig. 18-1). If these structures are ruptured from a flexion injury with translation of C1 anteriorly, the spinal cord can be impinged on between the posterior aspect of the odontoid process and the posterior rim of C1.[12] The spinal canal at C1 is large compared with other cervical levels and allows a greater degree of rotation and some displacement without spinal cord compromise. *Steel*[2] described the rule of thirds for the spinal canal at C1; the spinal canal at C1 is occupied equally by the spinal cord, odontoid, and a buffer space to prevent neurologic injury. Displacement of the atlas that exceeds the width of the odontoid may place the spinal cord at risk. Because of anatomical differences, the forces exerted at the craniocervical junction in children during trauma are significantly different from those in a similar event affecting adults. In the same manner, healing of these injuries may well differ significantly.

Clinical diagnosis of TAL disruption is difficult because there is often no visible external injury. A high index of suspicion and the use of modern neuroradiologic imaging remain the mainstay of diagnosis. Common red flags that should alert the physician to the possibility of TAL injury include a history of considerable trauma such as a motor vehicle accident, motor vehicle–pedestrian accident, sports-related neck trauma, horseback riding injuries, lack of vehicle restraint, fall from a height, penetrating injury, or suspicion

Fig. 18-1 Normal anatomical relationships of the atlantoaxial joint.

of child abuse.[6,13] Complaints of high cervical spine pain, particularly with nodding, muscle spasms, and headaches may present. Neurologic findings can vary from normal to some degree of quadriparesis. Permanent quadriplegia is highly unusual as severe spinal cord injury at this level usually results in death.

The cognitively impaired patient also presents another diagnostic dilemma. Signs of trauma to the head, face, or deformity such as torticollis may be present. In newborns and very young children, such signs may be absent. There is a higher incidence of spinal cord injury without radiographic

abnormality (SCIWORA) in the pediatric age group. One should beware of the small child who is cradling his or her head with his or her hands and complaining of occipital headache. Attention should be focused especially on the upper cervical spine in children younger than 8 years. A careful neurologic examination must be performed, including a cranial nerve assessment because high cervical injuries are commonly associated with multiple lower cranial nerve deficits.[6,13–15] Patients who survive this injury can often present with neurologic deficit ranging from complete high tetraplegia to incomplete injuries, such as mild paresis and brainstem

dysfunction or even death. Also, diffuse motor loss may occur if the pyramidal tract is affected. In some cases the diagnosis may be an incidental finding on flexion-extension plain radiography with no neurology. Noncontiguous spinal injury should be sought.

DIAGNOSTIC WORKUP

Diagnostic imaging studies of patients with suspected TAL injury should begin with plain cervical radiographic examination consisting of anteroposterior, open-mouth view, oblique, and lateral projections, and when appropriate, flexion and extension views (with the patient erect when possible). The pathologic characteristics of TAL injuries have been characterized using thin-section computerized tomographic scans and high-resolution magnetic resonance imaging (MRI).[15,16] Features suggesting loss of integrity of TAL are summarized in Table 18-1.

Roentgenograms demonstrate bilateral symmetrical overhang of the lateral masses of the atlas in relation to the axis, with an increase in the paraodontoid space on the open-mouth view (Fig. 18-2). Spence et al.[4] determined that if the lateral mass overhangs the articular surfaces of the axis more than 7 mm, a tear of the transverse ligament is likely, resulting in clinical C1-C2 instability. Increase of the atlantodental interval (ADI) signifies injury of the TAL. The ADI should be less than 5 mm in children younger than 9 years and less than 3 mm in adults. On the anteroposterior (open-mouth or odontoid) view of the craniocervical junction, the lateral masses of C1 should align exactly with the lateral margins of C2 when degenerative spurring is ignored (see Fig.18-2). If the lateral masses of C1 overhang articular surfaces of axis more than 7 mm, then transverse ligament is likely to be torn (Spence's rule).[4]

Plain radiographs alone may not to be sensitive enough to exclude injury to the TAL.[14,17] Normal relationships of the C1 lateral masses on open-mouth radiographs or a normal ADI on lateral cervical radiographs does not exclude injury to the transverse ligament, especially if the C1 ring is intact. It has been shown that by using only the radiographic criterion of

Fig. 18-2 C1-C2 overhang relationship illustrating Spence's rule.

more than 7 mm combined overlap of lateral masses on open-mouth radiograph, one would miss up to half the cases of TAL injury, and the ADI cut off of 3 mm was used, roughly one third of TAL injuries would be missed.[14,18] Also, flexion-extension radiographs are not advocated in patients with extensive fractures of C1 or C2 because of the fear of neurologic injury. Therefore, if radiographs are within normal limits or if they do not demonstrate any pathology in the presence a positive history and significant suspicion of injury, it is necessary to evaluate the patient further with computed tomography (CT) and or MRI.

CT, like plain radiography, indirectly assesses the probability of rupture of the transverse ligament. The CT studies of the cervical spine tend to demonstrate bony injuries to C1 to a large extent. The CT scan should be carefully checked for retropharyngeal hematoma, which suggests an acute injury, and for small flecks of bone avulsed off the lateral masses of C1, which may indicate avulsion of the ligament. CT scans demonstrate an avulsed tubercle of the lateral mass of C1 in Dickman type II injuries.[14,19]

Recently, it has been proposed that MRI is more sensitive in the diagnosis of TAL injuries than the rules of Spence. MRI can directly visualize the transverse ligament and therefore can add a different class of diagnostic information. Dickman et al.[16] recently demonstrated the MRI signal characteristics of the TAL in normal subjects and postmortem specimens using axial high-field MRI. They observed that gradient-echo MRI pulse sequences provided reliable visualization of the transverse ligament. The TAL has a homogenous low-signal intensity and extends behind the dens between the medial portions of the lateral masses of C1. The disrupted ligament appears as an anatomic discontinuity with high-signal intensity within the ligament and blood between the separated portions of the ligament. In summary, the most reliable ways of determining whether the transverse ligament is incompetent are with gradient-echo axial MRI,

TABLE **18-1**	Features Suggesting Loss of Integrity of Transverse Atlantal Ligament

Spence's rule: >7 mm combined overlap of lateral masses on open mouth
Bony avulsion fragments on CT suggest loss of ligamentous integrity
An increased ADI on the lateral >3 mm in adults >5 mm children implies rupture of the transverse atlantal ligament
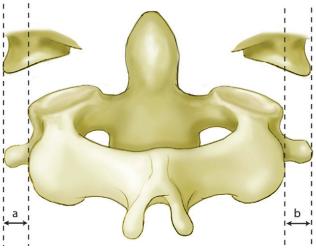MRI may demonstrate ligamentous rupture

ADI, Atlantodental interval; CT, computed tomography; MRI, magnetic resonance imaging.

which shows the anatomy of the ligament, or with flexion and extension radiographs, which measure whether C1-C2 anterior subluxation develops with mechanical loading.[14,16]

SURGICAL ANATOMY OF THE LIGAMENTOUS COMPLEX

It is important for surgeons to understand the anatomical and functional relationships of the bony articulations and ligaments of the craniocervical junction to allow them to predict the effect of injuries and surgical fixation on the stability of this region. The upper cervical spine consists of two uniquely shaped vertebrae, the atlas and the axis, intimately connected by a complex ligamentous network. The axis forms a unique articulation with the atlas. Their articulations allow rotation, accounting for nearly 50% of such motion in the cervical spine.[20] From the body of the axis, the odontoid process projects upward into the ring of the atlas. Under normal circumstances, the odontoid remains in contact with the anterior arch of the atlas, providing a point of action for the osseous and ligamentous structures that limits atlantoaxial motion.

The transverse ligament of the atlas is a thick, strong band that which arches across the ring of the atlas and retains the odontoid process in contact with the anterior arch. It is concave in front, convex behind, broader and thicker in the middle than at the ends, and firmly attached on either side to a small tubercle on the medial surface of the lateral mass of the atlas. It joins with the posterior facet of the odontoid. As it crosses the odontoid process, a small fasciculus (superior crus) is prolonged upward, and another (inferior crus) downward from the superficial or posterior fibers of the ligament. The former is attached to the basilar part of the occipital bone, in close relation with the membrana tectoria; the latter is fixed to the posterior surface of the body of the axis; hence, the whole ligament is named the cruciate ligament of the atlas. The transverse ligament is the primary restraint to anterior atlantoaxial subluxation. The accessory ligaments similarly arise from the lateral masses and insert into the base of the odontoid. The secondary stabilizers consist of the apical, atlantodens and alar ligaments. The apical ligament arises from the anterior rim of the foramen magnum and inserts into the tip of the odontoid. The alar ligaments arise from the medial aspect of the occipital condyles and the lateral masses of the atlas. They insert broadly into the lateral portion of the odontoid. They restrict contralateral rotation and lateral bending of the atlas on the axis.[21,22]

BIOMECHANICS OF TAL INJURY

The characteristics of the TAL have been described in recent times.[4,17,23–26] The TAL is inelastic and therefore excessive load results in ligamentous disruption rather than stretch. If the ligamentous substance is completely disrupted, it typically does not heal. When torn, permanent anterior atlantoaxial instability exists. The transverse ligament may be avulsed with a bony

fragment from the lateral mass on either side, or it may rupture in its mid substance. Mid-substance tears are far less common than bony avulsions. In burst fractures, rupture of the transverse ligament occurs as a result of tension secondary to displacement of the lateral masses of the atlas; however, the apical and alar ligaments are spared, in distinction to a transverse ligament rupture from a severe flexion injury. Additionally, the anterior and posterior C1-C2 facet capsules are spared in the burst fracture, resulting in intact secondary flexion restraints. It has been estimated that a force of about 85 kg is required to rupture the transverse ligament.[4,14,17,23] An anterior shift of C1 on C2 of more than 3 to 5 mm implies injury to transverse ligament, whereas a shift of more than 5 mm implies injury to alar and transverse ligaments.[4]

Spence et al.,[4] in 1970, reported their findings of a study of the mechanism of atlas fracture and potential rupture of the TAL. Using 10 cadaveric specimens, the authors studied the application of force required to fracture C1 and to rupture the transverse ligament (range, 38–104 kg; mean, 58 kg). The sum of the excursion of the C1 lateral masses over the C2 lateral masses after traumatic injury ranged from 4.8 to 7.6 mm (mean, 6.3 mm). The authors concluded that if the sum of lateral mass displacement (LMD) of C1 over C2 on the anteroposterior radiographic image is more than 6.9 mm, then the TAL is "probably torn." In a follow-up clinical and biomechanical study, Fielding et al.[23] confirmed these findings. They experimentally produced a transverse ligament defect with intact alar and apical ligaments resulting in a maximal translation of 5 mm in the ADI. Displacement of more than 7 mm was associated with loss of integrity of the alar ligament and tectorial membrane. They further concluded that in severe flexion injuries with transverse ligament ruptures associated with additional injury to the alar ligament and facet capsules, gross clinical instability can result. These two studies, completed before the era of MRI, are the basis for the widely quoted "rule of Spence" (i.e., >6.9 mm LMD = TAL disruption) offered to assist in the management of patients with isolated atlas fractures.

Heller et al.[18] reported their observations on 35 open-mouth odontoid films using calibration markings to assess radiographic magnification. They found an 18% magnification factor on open-mouth odontoid-view x-rays. Applying this information to the evaluation of atlas burst fractures by means of the rules of Spence suggests that the sum of the LMD measurements indicating atlantal transverse ligament disruption should be increased from 6.9 to 8.1 mm. This study pointed out the difficulty in using plain radiographic measurements to assess the integrity of the TAL after acute traumatic atlas fracture.

INJURY CLASSIFICATION AND TREATMENT OPTIONS

Injuries to the TAL have been classified according to whether the ligament and its insertions are primarily involved or whether fractures of the osseous lateral masses are present,

without disruption of the ligament. Dickman et al.[14] described two types of isolated TAL injuries they identified on MRI. In type I, the substance of the ligament is injured without associated fracture of the atlas. Type II injuries involves an avulsion fracture of the atlas at the insertion of the TAL. They suggested that the two types of injuries had distinct clinical characteristics and treatment outcomes. They, however, did not include burst fractures of C1 (Jefferson's fracture), which accounts for a majority of clinically significant TAL injuries. They further classified these injuries into A or B subgroups as shown in Table 18-2 and Figure 18-3. Type IA includes all mid-substance ligament tears, whereas IB includes osteoperiosteal disruptions of the TAL. Type IIA injuries typically had an associated lateral mass fracture of C1 along with a bony avulsion of the TAL. Type IIB injuries are associated with tubercle avulsion without comminuted fractures of the involved C1 lateral mass.

 Current treatment standards for TAL injuries are based on limited data about natural history and results of the different surgical options.[4,14,27–34] In general, patients with significant instability primarily resulting from ligamentous injury need to be surgically stabilized and fused. In the occasional patient with significant instability and a bony avulsion injury with an intact ligamentous complex, nonsurgical treatment may be attempted. Previous reports on the treatment of this condition have shown that treatment depends on the degree of initial displacement and identification of any simultaneous fractures.[4] Generally speaking, if the ADI is less than or equal to 5 mm in a neurologically intact patient, rigid external immobilization is sufficient initially. For an ADI more than 5 mm, nonsurgical treatment including halo immobilization has generally yielded poor results except for selected cases when a bony avulsion can be documented on CT. If a transverse ligament disruption exists with an atlas fracture, C1-C2 fusion is required to restore stability to the C1-C2 segment.

Fielding et al.[31] recommended the initial use of head-halter traction until muscle spasm resolves, followed by a brace support. They also concluded that, if flexion-extension roentgenograms subsequently demonstrated significant instability, C1-C2 fusion should be indicated. Fielding et al.[31] stated that atlantoaxial fusion may be the "conservative" treatment for this lesion. They recommended posterior C1-C2 fusion using wire fixation and an iliac bone graft.

Classification of Injuries to the Transverse Atlantal Ligament

I. Ligament rupture
Midportion (IA)
Periosteal insertion (IB)
II. Avulsion of insertion of the transverse ligament into lateral mass
Involving a comminuted C1 lateral mass (IIA)
Avulsion of the tubercle from an intact lateral mass (IIB)

Spence et al.[4] based treatment of TAL injuries with associated burst and lateral mass fractures on the amount of LMD or instability, determined by open-mouth radiograph. Minimally displaced fractures (<7 mm total displacement) or significantly displaced fractures (7 mm) correspond to the integrity of the transverse ligament. These fractures are considered stable when the combined lateral overhang of the atlas measures less than 7 mm. They stated that nondisplaced and minimally displaced fractures can be immobilized in a collar; displaced fractures require more definitive surgical treatment.

Dickman et al.[14] in their review of 39 cases of TAL injuries concluded that type II injuries with associated bony injury had a greater chance of healing without surgical intervention. None of the type I injuries healed spontaneously, and all required C1-C2 fusion to restore permanent atlanto-axial stability. In their study, C1-C2 stabilization involved the use of wires or cables and autologous graft for posterior fusion. About 30% of these patients required transarticular fixation of C1-C2. Patients with type II injuries were initially managed nonoperatively using a Philadelphia collar or a Halo brace for 13 weeks, with 74% of them going on to heal. They further showed that a small subgroup of their patients with tubercle avulsions (type IIB injuries) may have a particularly high risk for nonunion.

Evidence of healing and successful treatment have been extrapolated from findings of reduced abnormal alignment and predental interval between the atlas and the axis, coupled with serial clinical evaluations demonstrating full neck movement and the absence of neurologic symptoms. Patrick et al.[35] reported the results obtained in three pediatric patients, with TAL avulsion injury with tubercular fracture, demonstrating healing clinically and radiologically with the use of nonsurgical external immobilization therapy. On CT, healing was evidenced by ossification and "reattachment" of the osseous fragments. This is the first evidence reported in the English-language literature to confirm radiologic healing in this condition in childhood.

Surgical technique for C1-C2 fusion can vary. Older literature describes posterior surgical stabilization of C1-C2 can be done through the use of wire techniques. With the Gallie type of wire fixation method, a posterior vector force on C1 is applied, bringing the C1-C2 relationship into a reduced position. With an intact dense, the dens prevents C1 from being pulled too far posteriorly, and therefore overreduction is impossible. If a bone block techniqu wire technique such as the Brooks fusion is performed, the ring of C1 may redisplace anteriorly. If the posterior arch of C1 is fractured, a halo vest should be used for 8 to 12 weeks to allow healing of the posterior arch before proceeding with a standard C1-C2 posterior arthrodesis. Levine and Edwards[33] found an average loss of correction of 4 mm after bone block techniques and 1 mm after Gallie wiring. Postoperative displacement can still occur even with halo immobilization postoperatively. More recent literature describes the more rigid screw fixation methods.
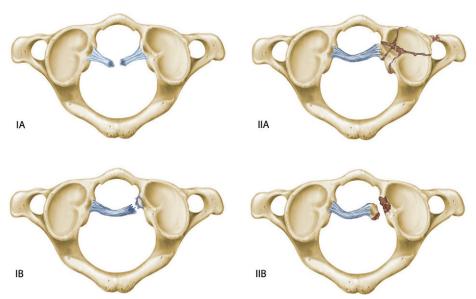
Fig. 18-3 Classification of TAL injuries. Type I injuries disrupt the ligament substance in its midportion *(IA)* or at its periosteal insertion *(IB)*. Type II injuries disconnect the tubercle for insertion of the transverse ligament from the C1 lateral mass involving a comminuted C1 lateral mass *(IIA)* or avulsing the tubercle from an intact lateral mass *(IIB)*.

This can be achieved with transarticular screw fixation or C1 lateral mass screws couple with C2 pedicle/isthmus screws.[27–34] Transarticular screw fixation brings can be associated with vertebral artery injury and therefore careful preoperative imaging (usually CT scans) are necessary to determine whether the anatomy will allow this method. C1 lateral mass screw fixation can reduce risk of vertebral artery injury; however, the exposure required can be associated with heavy venous plexus bleeding. With these newer fixation methods halo immobilization is generally not required and results in a very high rate of union.

CONCLUSION

In summary, the treatment of injuries to the upper cervical spine is based on the extent of injury to both the ligaments and the bone structures involved. Disruptions of the substance of the TAL (type I injuries) are incapable of healing and should be treated with early surgery for internal fixation. Surgery for internal fixation of type II injuries is reserved for individuals who develop nonunion with persistent instability after a reasonable duration of halo immobilization (12–16 weeks). Also, close monitoring is mandatory to detect patients who require delayed operative treatment.

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Atlantoaxial Rotatory Deformities

INTRODUCTION

Atlantoaxial rotatory instability (AARI) refers to pathologic motion that occurs at the C1- C2 articulation. It is an uncommon condition that occurs predominantly in children. The discussion of atlantoaxial rotatory deformities focuses on a continuum of two entities, AARI and atlantoaxial rotatory fixation (AARF). AARI is considered a decoupling of the normal motion of the C1-C2 articulation. AARF is the pathologic interlocking of the C1-C2 joint. With the inability to accurately assess the biomechanical interrelationship of this joint, practitioners have been unable to precisely define these pathologic conditions. Indeed, many a physician has been befuddled by plain radiographs in patients who present with torticollis. The abnormality, in many cases, is indistinguishable from normal rotation. Even static computed tomography (CT) imaging in evaluating patients with torticollis in many cases is ambiguous. Further clarification of the complex interrelationship of the movement of C1 on C2 throughout rotation will lead to more accurate diagnosis and effective management.

Most often, this entity follows an upper respiratory tract infection, although it has been identified after tonsillectomy and even minimal trauma.[1,2] In adults, its occurrence is usually associated with major trauma, although it has been described in patients with inflammatory conditions of the spine such as rheumatoid arthritis and ankylosing spondylitis.

The understanding of the pathoetiology of rotatory atlantoaxial instability continues to evolve. A dynamic complex relationship exists between the C1 and C2 segment. During maximal rotation, the atlas may become fixated on the axis. This may be caused by ligamentous laxity of the alar and transverse ligaments as well as the C1-C2 lateral mass joint capsules. The normal laxity present in children most likely predisposes them to this condition in the presence of proximate inflammatory changes or mild trauma.

Both adults and children with rotatory C1-C2 instability present with the classic signs of torticollis (Fig. 19-1). There is a loss of motion in the cervical spine in all planes, and rotation generally does not cross the midline on the side that the head is tilted. Pain is not a ubiquitous complaint.

Radiographic evaluation may be difficult, especially in severe cases. Because of marked rotation and tilt, lateral cervical spine films are difficult to interpret secondary to overlap of the mastoid process. Multiplanar CT is the imaging study of choice to visualize the spine in both the sagittal and coronal plane.

The mainstay of treatment remains nonoperative. Indeed, many patients with a milder form of this condition probably do not receive medical attention at all. For patients that do present to a physician, a soft cervical collar and medical therapy is commonly used and occasionally traction may be required in more refractory cases. Surgery is reserved for patients with fixed deformities and is more commonly performed in the adult patient with a traumatic or inflammatory etiology.

Torticollis is a common childhood occurrence that is only rarely associated with AARI. Torticollis most commonly involves a soft tissue abnormality of the neck, specifically the sternocleidomastoid muscle.

ANATOMY

The bony anatomy of the C1-C2 articulation allows for maximum flexibility. Indeed, of the approximately 160 degrees of possible rotation of the cervical spine, the atlantoaxial joint provides upwards of 80 degrees. A unique feature of the atlantoaxial articulation is the horizontally-oriented facets, which rely almost entirely on ligaments for stability. The alar ligaments function as a check against excessive rotation.[3]

Although the primary motion at C1-C2 is rotation, there is some anterior-posterior motion and tilt, as well as a horizontal slide (translation), that occurs with rotation. The understanding of the complexity of motion of this joint has recently been elucidated.[4] During the initiation of C1 rotation (0–23 degrees), the atlas moves alone. When C1 rotates from 24 to 65 degrees, C1 and C2 move together with C1

Fig. 19-1 A child with typical cock robin deformity consistent with torticollis. Note the head is tilted and rotated to the contralateral side. Spasm of the sterno-cleidomastoid muscle is present on the long (left) side.

moving at a slightly more rapid rate. Upwards of 65 degrees of rotation, C1 and C2 move in unison with a fixed maximum separation of 43 degrees.

It must be remembered that stability of the upper cervical spine is almost entirely dependent on the surrounding soft tissue ligamentous constraints. The transverse ligament is the primary stabilizing force. Any laxity or incompetence of the transverse atlantal ligament, which fixes the odontoid in the anterior one third of the spinal canal at C1-C2, may cause impingement on the space available for the cord (SAC). The canal at this level is quite capacious, consequently neural deficits are rare.

Physiologic differences are apparent between the child and the adult. Specifically, children younger than 8 years have increased cervical motion secondary to ligamentous laxity, relative muscle weakness, and incomplete ossification of the cartilaginous elements of the pediatric cervical spine.[5] Incomplete ossification in the cervical spine in children accounts for differences in measurements of certain relationships, including the basion-odontoid interval and the atlanto-odontoid interval.[6]

Classification of atlantoaxial rotatory subluxation is predicated on the direction and degree of displacement of this articulation.[7] In type I atlantoaxial fixation, simple rotatory displacement with no anterior shift of C1 on C2 is present. This is the most common type in children. Type II notes anterior shift of 5 mm or less in addition to rotatory displacement. This lesion is fairly common with the potential for neural injury. Type III and IV deformities are rare. Type III injury exhibits anterior shift greater than 5 mm with significant obliteration of the SAC. This type is most commonly associated with neurologic involvement of either the spinal cord or proximate cranial nerves. Type IV deformities are associated with a posterior shift of C1 on C2 (Fig. 19-2).

White and Panjabi[8] have also proposed a classification system. They recognized three entities. Type A is similar to the Fielding type I fixation with odontoid pivot only. Type B is defined by posterior displacement similar to Fielding type IV fixation. Type C is defined by anterior displacement C1 on C2, similar to Fielding type II and III fixation.

PATHOGENESIS

The true etiology of this entity is unknown. AARI has been identified in numerous physiologic processes. Pharyngitis, with involvement of the cervical lymph nodes, is a common cause of AARI in children. Additionally, it has been observed following surgical procedures. Following surgery such as tonsillectomy, inflammation and local edema in the retropharyngeal space may predispose for C1-C2 subluxation. It is hypothesized that local swelling secondary to an inflammatory process leads to softening and possible disruption of the alar and transverse ligaments. Venous and lymphatic connections between the superior retropharyngeal region and the upper cervical spine allows for spread of local hyperemia.[9] Evidence of meniscal infolding of synovium has also been identified in the lateral atlantoaxial joints of children.[10] The excessive motion allowed at this articulation by the surrounding soft tissue laxity may allow synovium to become entrapped in the C1-C2 articulation, which leads to a fixed deformity.

An association with connective tissue disorders, such as Marfan syndrome, has been reported.[11] In two patients, AARI was diagnosed following pectus excavatum repair. The cervical bony and ligamentous abnormalities seen in patients with Marfan syndrome may increase their risk for AARI. Special attention to intubation and positioning, both intraoperatively and postoperatively, may be necessary.

In addition, atlantoaxial rotatory subluxation can occur in children after trivial trauma. AARI may occur secondary to a fracture, most commonly of the odontoid or an upper cervical spine ligamentous injury.[12]

Clinically, patients will present with head tilt to one side with rotation to the opposite side. The sternocleidomastoid on the opposite side of the head tilt (the long side) is usually in spasm secondary to its attempt to correct the deformity. Facial flattening, although rare, may be seen with fixed longstanding deformities in children. It has been known to correct after treatment.

Cranial nerve injury, although uncommon, must be looked for, especially in adults. Cranial nerve abnormalities that have been identified include IV, V, VI, IX, and X.
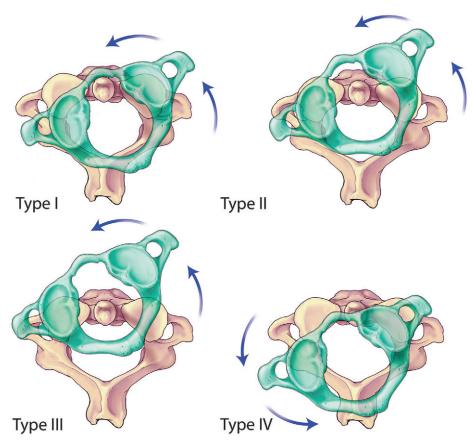
Fig. 19-2 The Fielding classification system for AARI.

Tongue paralysis resulting from hypoglossal nerve injury (CN XII) has also been reported.[13]

The proposed mechanism for cranial nerve involvement is mechanical traction or, more likely, vascular insufficiency.

DIFFERENTIAL DIAGNOSIS

In the child, other inflammatory conditions, autoimmune conditions such as juvenile arthritis, or an acute calcified cervical disk herniation may be the cause of AARI. The physician must also consider congenital muscular torticollis (wry neck). This entity presents usually several weeks after birth. Soft tissue abnormalities or tumors of the sternocleidomastoid muscle such as cystic hygroma, branchial cleft cysts and thyroid teratomas may also cause AARI. Skeletal dysplasia, Klippel-Feil syndrome, and Down's syndrome associated with C1-C2 instability may present as torticollis that mimics this entity.[14]

Trauma in the presence of comorbidities such as ankylosing spondylitis is the usual presentation of AARI in the adult. Less commonly, an infectious process such as tuberculosis or typhoid may be the underlying etiology. More rarely, spinal cord tumors, chordoma, posterior fossa tumors, syringomyelia, and bulbar palsies may be associated with a clinical torticollis. Generally, in these conditions, additional neurologic findings such as proximal weakness or the development of long tract signs confirm a more malignant process. Vision or hearing difficulty can also present with torticollis in the adult, which again emphasizes the necessity for a complete physical examination to identify the multitude of potential etiologies.[15]

DIAGNOSIS

Confusion has existed in defining AARI, most probably secondary to the difficulty in delineating the anatomy radiographically. A pathologically fixed state may be indistinguishable from normal rotation on a static film. Many consider AARI a fixed interlocking of C1 on C2. Others feel that AARF represents the final stage of a continuum. Thus, there has been great difficulty in defining and identifying this entity. By formulating a clear understanding of normal biomechanics of the C1-C2 articulation we can now move forward in accurately defining criteria for the diagnosis of AARI. The definition of AARI is now predicated on identifying abnormal rather than absent motion.

Accurate diagnosis is predicated by the history and clinical findings as radiographic findings are nonspecific and may occur secondary to other causes of torticollis (Fig. 19-3). Nevertheless, all patients with torticollis should undergo roentgenographic evaluation to exclude fracture or other bony

Fig. 19-3 A true lateral film depicting abnormal anatomic position of C1 on C2.

Fig. 19-4 Proper positioning of the patient for a true lateral cervical x-ray.
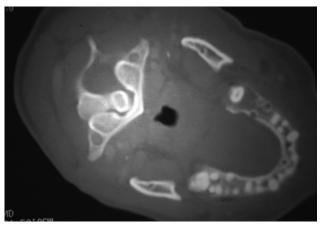
Fig. 19-5 CT scan delineating the pathoanatomy of AARF.

abnormality. Restricted motion and fixed head position make obtaining a lateral radiograph difficult. It should be remembered that the atlas moves with the occiput. By positioning the x-ray beam to obtain a lateral of the skull, a satisfactory view of the upper cervical spine can be obtained (Fig 19-4). A similar appearance of pathologic rotation may be obtained in a normal child whose head is rotated with that of a patient with AARI. Cervical rotation makes interpretation of plain radiographs difficult. Open-mouth anteroposterior (AP) radiographs are usually inadequate. Some have suggested open-mouth films with the patient rotating the head to each side.[16] A CT scan through the occipital cervical junction with both left and right rotation may clarify interpretation of pathology (Fig. 19-5).[17] In patients with AARI, the C1-C2 facets may appear locked despite left and right rotation (Fig. 19-6).[2]

Magnetic resonance (MR) imaging accurately depicts the anatomical integrity of the transverse ligament. This may be assistive in both the diagnosis and surgical decision making.[18]

TREATMENT

Acute torticollis itself probably resolves spontaneously in most cases. However, most patients with AARI probably seek treatment. Excellent results are obtained with conservative
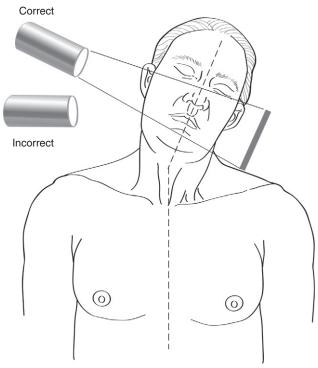
measures in most children. For individuals with symptoms less than 1 week, immobilization with a soft cervical collar and anti-inflammatory medications usually suffice. Complete and pain-free motion of the neck should be observed before discontinuation of external immobilization. In patients in whom reduction fails to occur spontaneously or in patients who have a history of AARI for greater than 1 week, cervical traction is recommended. In most cases head halter traction is preferable. Immobilization in a rigid orthoses such as a Philadelphia collar or a sternal occipital mastoid
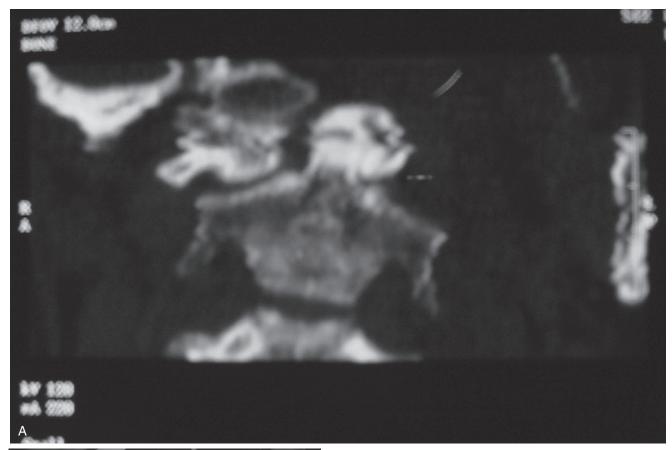
Fig. 19-6 *A,* A sagittal reconstruction of a patient with AARI. *B,* A three-dimensional (3D) reconstruction of a patient with AARI.

immobilization (SOMI) brace for a minimum of 4 weeks is recommended.

In patients with AARI for greater than 1 month, successful results with cervical traction are guarded. It is in this group of patients in whom surgical treatment should be considered. Similarly, in patients who have a loss of reduction after prior treatment usually require surgical stabilization.

For patients in whom instability persists, several surgical approaches have been advocated. Fielding recommended

C1-C2 arthrodesis. Transoral facetectomy followed by reduction and then occipitocervical arthrodesis has also been described.[19] The authors favor traction followed by open reduction using C1 lateral mass and C2 pedicle or isthmus screws or C2-C1 transarticular screws.

Adults diagnosed with this disorder in its chronic stage or associated with advanced connective tissue disorders do not fare as well with nonoperative treatment. Although exceedingly rare following acute trauma, with only four cases

reported in the literature, Wise et al.[20] noted that following a satisfactory closed reduction, a period of immobilization is adequate without the need for surgery.

The majority of these patients require operative stabilization for relief of symptoms. In patients with severe deformity, closed skeletal traction reduction is recommended. However, reduction in the presence of significant AARI may result in neurologic compromise.[12,21]

Several closed traction techniques in the adult have been advocated. Axial traction using Gardner-Wells tongs is recommended in the adult patient. The patient should be slightly sedated. A gentle reduction maneuver using manual rotation can be performed following axial traction application. Levine and Edwards[22] additionally anesthetized the oropharynx. They then recommended applying gentle pressure anteriorly over the C1 and C2 step off.

During a reduction maneuver, the patient should be monitored for additional neurologic injury especially to the cranial nerves. Following satisfactory reduction in the acute setting, immobilization using a rigid orthosis is recommended for approximately 3 months. Traction reduction in the chronic state should be followed by surgical stabilization.

In summary, AARI is an uncommon dissociation of the normal biomechanical and physiologic relationship of the C1-C2 joint complex. If because of localized inflammation without significant soft tissue attenuation or destruction, prompt recognition and institution of treatment often results in amelioration of this disorder. The physician must exercise diligence in obtaining satisfactory imaging studies to properly recognize this condition. Following its identification and treatment, close follow-up is necessary to avoid delay in detecting loss of reduction in patients treated nonoperatively.

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C H A P T E R

20

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Odontoid Fractures in Pediatric and Adult Patients

Odontoid fractures are commonly encountered in spinal trauma as they comprise approximately 10% to 20% of all cervical spine injuries.[1–3] Although a large percentage of odontoid injuries occurs in older adults, these injuries can occur in individuals of any age. In children younger than 7 years, a synchondrosis (cartilaginous endplate) exists just below the base of the odontoid process in the upper portion of the body of C2. The synchondrosis is prone to injury with hyperflexion trauma. After this synchondrosis closes, the base of the odontoid is strengthened, and high-energy trauma is required to produce this fracture in healthy, nonosteoporotic adults. In elderly patients with osteoporosis, however, the odontoid is susceptible to fracture with even mild trauma, such as ground-level falls. In this chapter, we review injuries to the odontoid process in adult and pediatric patients.

Because of the unique anatomy required to achieve the specialized function at the C1-C2 joint, odontoid fractures are serious injuries that result in instability and carry the potential of catastrophic spinal cord injury. The C1 vertebra is a transitional vertebra between the skull and spine with a condylar joint articulation that primarily allows flexion and extension. In contrast, the C1-C2 articulation is designed to facilitate rotation, with relatively flat joint surfaces that slope gently upward from lateral to medial and loose capsular ligaments to allow significant rotatory motion. The looping course of the vertebral artery and the very generous spinal canal dimensions at this level also facilitate the 45 degrees of rotation to either side that can occur at this joint—about half the rotation of the head. Only an intact odontoid process contained within the anterior portion of the ring of C1 by the strong transverse portion of the cruciate ligament prevents translation, which could cause cord compression resulting in quadriparesis or death.

The presence of an intact odontoid process and transverse ligament is therefore critical to the patient's well-being.

Patients with injuries to these structures are inherently unstable and at risk for further injury. Treatment is initially directed to providing external stabilization and then toward measures to restore stability permanently, either by achieving healing of an odontoid fracture or by fusing C1 to C2.

ODONTOID INJURIES IN CHILDREN

The axis develops from five primary ossification centers (one within the body, two within the odontoid, and one in each posterior arch) (Figs. 20-1, *A* and *B*). In the developing spine, the primary ossification centers are separated by a cartilaginous endplate, called a synchondrosis. As the developing spine matures, these synchondroses ossify and close. In the axis, the two ossification centers in the odontoid are the first to close, about the time of birth. The subdental synchondrosis, between the odontoid and the body (Fig. 20-1, *C*), is the last to close. This process starts around 4 years of age and is completed in most children by the age of 7. In approximately one third of adults, some cartilage remnants persist in the area of the previous subdental synchondrosis.[4,5]

The large size of a child's head in relation to his or her body creates a fulcrum at the craniocervical junction that, along with the area of weakness at the subdental synchondrosis, increases the likelihood for odontoid injury with trauma. One common scenario is a toddler in a forward-facing car seat involved in a motor vehicle accident. As a result of the relatively large size of the child's head and underdeveloped neck musculature, the head continues forward with the momentum of impact while the body remains fixed, resulting in a high-energy hyperflexion injury. Thus, in young children, most cervical spine injuries occur in the upper cervical spine with the axis being the most commonly injured vertebra in young children. Subdental synchondrosis fractures are one of the more common fractures in children younger than 7 years.[6,7]

CLINICAL PRESENTATION

Odontoid synchondrosis fractures most commonly present with neck pain after trauma. In young, noncommunicative children, it can be difficult to determine the cause of pain or
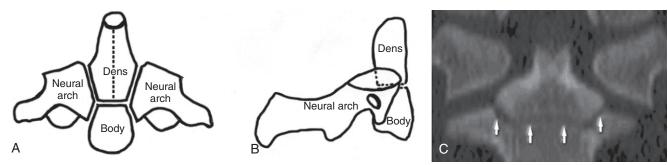
Fig. 20-1 C2 developmental anatomy. The axis develops from five primary ossification centers: one on each lateral half of the odontoid process, one within the body, and one in each neural arch. These ossification centers are separated by a cartilaginous endplate, called a synchondrosis. Anteroposterior *(A)* and lateral *(B)* illustrations of the axis show the ossification centers and synchondroses between these areas. *C,* The subdental synchondrosis, as shown in the coronal CT reconstruction in a normal 4-year-old child, is the last to close and thus represents a potential area of weakness in traumatic injuries of young children.

irritability. If the child is splinting or preventing head and neck movements, cervical spine injury should be strongly considered, with specific attention to rule out odontoid synchondrosis fractures in children younger than 7 years. Although most patients do not suffer neurologic injury with odontoid synchondrosis fractures, the incidence of spinal cord injury in these young children appears to be greater than that in adults with odontoid fractures. In a combined series of published odontoid synchondrosis fractures, we found a 27% (15 of 55) incidence of spinal cord injury in children with odontoid synchondrosis fractures.[8] Complete spinal cord injury was noted in 11 patients and 8 patients had spinal cord injuries at the cervicothoracic junction (Fig. 20-2).

We theorize that the high incidence of cervicothoracic junction spinal cord injuries, remote from the fracture site, is because of a stretch injury to the spinal cord associated with the hyperflexion mechanism.

RADIOGRAPHIC EVALUATION

Odontoid synchondrosis fractures are most commonly diagnosed with plain lateral radiographs, which typically show anterior displacement or angulation of the odontoid. In the combined series we reviewed,[8] the odontoid was anteriorly angulated or displaced in 94% of cases. This angulation may be subtle and the diagnosis of this fracture can
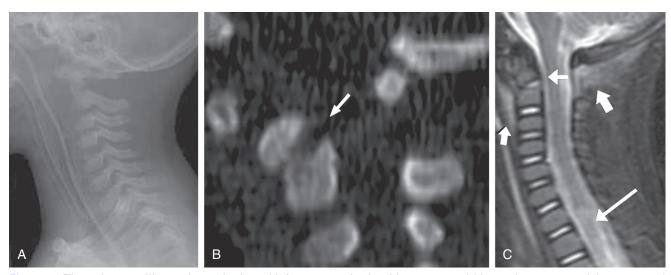

Fig. 20-2 These images illustrating spinal cord injury were obtained in a 2-year-old boy who was struck by a car and was noted to have no motor function in legs on evaluation. *A,* Although the lateral plain radiograph was initially interpreted as normal, there appeared to be anterior angulation of the odontoid on closer evaluation. *B,* CT sagittal reconstructions showed angulation of the odontoid and widening of the posterior aspect of the synchondrosis *(arrow). C,* T2-weighted sagittal MRI showed spinal cord edema *(long white arrow)* at the cervicothoracic junction with high-signal at the odontoid synchondrosis *(short white arrow)* and soft tissues *(thick grey arrows)* anteriorly and posteriorly at C1-C2.

be easily missed on plain films if the odontoid is not examined carefully. In our series, a delay in diagnosis was noted in 8 of 55 cases.

Computed tomography (CT) and magnetic resonance imaging (MRI) can both be used to confirm the diagnosis of odontoid synchondrosis fractures. We prefer to use MRI to evaluate cervical spine injuries in young children because this modality reveals ligamentous injuries, which are more common than fractures in children. MRI also does not put the patient at risk of radiation exposure. In addition to showing angulation of the odontoid, MRI may also show abnormally high signal on short tau inversion recovery (STIR) sequences within the subdental synchondrosis and possibly high signal in the anterior and posterior soft tissues at the level of injury. If neurologic deficits that could be attributable to spinal cord injury are present, an MRI of the cervical spine should be performed to also exclude other causes of the neurologic deficits. Thin-cut axial CT scanning with two-dimensional reconstruction in the coronal and sagittal planes shows the synchondrosis well. Widening of the synchondrosis (usually posteriorly) is suggestive of synchondrosis fracture.[9]

TREATMENT OF ODONTOID SYNCHONDROSIS FRACTURES

Conservative treatment of synchondrosis fractures with external orthosis is recommended as the initial treatment in most cases. Halo vest immobilization or Minerva brace is most commonly used, with greater than 90% of these fractures healing with external immobilization.[8] Some authors have used early surgical stabilization in children with significant neurologic deficits as a means to provide for earlier mobilization and rehabilitation.[10] In most situations, however, surgery has been reserved for patients who fail conservative therapy, namely those who experience resubluxation despite external stabilization or those who have nonunion despite 3 to 6 months of conservative treatment.[11] We use bone bridging across the synchondrosis or lack of motion on flexion-extension images at this level to assess the healing.

Posterior C1-C2 arthrodesis has been used in most cases when patients have failed conservative therapy. Some authors have used posterior wiring and bone grafting alone (either Sonntag or Brooks-Gallie fusion), but we believe that instrumentation with atlantoaxial transarticular screws provides more immediate stability and improves fusion rates without the need for supplemental external orthosis after surgery in most cases (Fig. 20-3).[11] Fusion appears to be well tolerated by these young children, but the long-term effects of atlantoaxial fusion in these patients are unknown.[12] Blauth et al.[13] reported success in two cases of surgical stabilization with motion-sparing techniques. In one case, they successfully used an odontoid screw to treat an acute synchondrosis fracture. In another case of delayed nonunion of a synchondrosis fracture, they used a temporary posterior atlantoaxial wiring technique in conjunction with anterior bone grafting in the synchondrosis. After successful bone healing across the synchondrosis was achieved, the authors removed the posterior wiring and reported that normal motion was maintained.
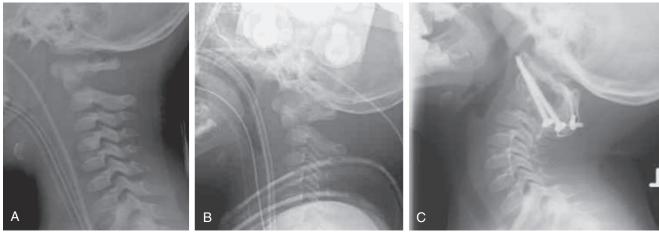

Fig. 20-3 Images of a 22-month-old girl who was injured in a motor vehicle accident while restrained in a forward-facing child car seat. No neurologic deficits were noted on examination, but the child was very irritable. *A,* Lateral radiograph showed anterior angulation of the odontoid process consistent with an odontoid synchondrosis fracture. The patient was treated with reduction with the aid of fluoroscopy to confirm good alignment and placed in a halo vest orthosis. *B,* Follow-up lateral radiograph obtained 1 week after halo placement showed resubluxation of her odontoid synchondrosis fracture. *C,* The patient was subsequently treated with surgical stabilization with posterior C1-C2 arthrodesis with atlantoaxial transarticular screws (postoperative lateral radiograph is shown). She went on to attain fusion without complication.

ADULT ODONTOID INJURIES

Odontoid fractures are much more common in adults than in pediatric patients. The mean age reported in published surgical series is between 41 and 67 years, with a bimodal distribution.[1,14–17] In young adults, which comprise the first peak, odontoid fractures are usually a result of high-energy trauma, whereas in the elderly, odontoid fractures are commonly seen with minor trauma, such as ground-level falls.[1,18] In people older than 70 years, odontoid fractures are the most common cervical spine fracture.[19–21]

CLINICAL PRESENTATION

Neck pain is the most common clinical finding.[22] In multitrauma patients, this fracture is often associated with other injuries, including closed-head injuries, which can obscure the clinical examination or make it difficult.[1] Spinal cord injuries are not common with odontoid fractures, occurring in only 10% to 16% of patients in published clinical series; however, spinal cord injury at this level in the upper cord may result in death before radiographic evaluation, which may result in under-reporting of spinal cord injury.[15,17] Nineteen percent of patients have other cervical spine fractures associated with their odontoid fracture.[17]

RADIOGRAPHIC EVALUATION

Radiographic evaluation of the cervical spine in trauma patients often begins with plain three-view (anteroposterior, lateral, and open-mouth odontoid) radiographs. With advancements in high-speed CT imaging; however, plain radiographs of the cervical spine are occasionally omitted as part of the initial workup for multitrauma patients because of the speed and ease of screening multiple areas of the body with CT. When relying on three-view plain films, it is important to consider that the craniocervical junction can be obscured by surrounding anatomy and, as a result, these plain radiographs have been reported to be only 65% to 95% sensitive for detecting axis fractures.[3,23–25] In patients in whom there is a high clinical suspicion for cervical spine fracture, fine-cut CT scans are the study of choice for evaluating bony injury to the cervical spine. With CT studies, it is very important to evaluate the sagittal and coronal reconstructions of the axial CT images as odontoid fractures may occur in the axial plain and may be missed if one relies on axial images alone.

At our institution, MRI is used sparingly for the evaluation of odontoid fractures in adults and is usually reserved for patients with neurologic deficits. Some authors have recommended MRI evaluation of all odontoid fractures to evaluate the integrity of the transverse atlantal ligament as a means of guiding management.[26,27] Greene et al.[27] reported transverse ligament disruption in 10% of odontoid fracture patients and recommended early posterior arthrodesis for stabilization of patients with MRI evidence of transverse ligament

injury and an odontoid fracture. Although transverse ligament disruption is a potential concern, the incidence of transverse ligament disruption with odontoid fracture, as noted on delayed flexion-extension radiographs, is much lower in our experience and does not require early MRI evaluation, although this may be optionally obtained.

Odontoid fractures are most commonly characterized and described based on the radiographic features observed using the Anderson and D'Alonzo classification system.[14] In 1974, Anderson and D'Alonzo classified odontoid fractures into three types (Fig. 20-4) on the basis of the anatomic location of the fracture on the odontoid process as visualized on plain radiographs. Although modifications have been proposed, the Anderson and D'Alonzo classification is still the classification most commonly used to characterize odontoid fractures because of its simplicity and reliability as a predictor of outcome.

Anderson and D'Alonzo type I fractures involve the tip of the odontoid and are thought to be an avulsion fracture of the apical ligament. Type I fractures are extremely rare (approximately 1% of all odontoid fractures) and are generally thought to be stable unless they are associated with atlanto-occipital dislocation.[1,28] Type II fractures occur at the junction of the odontoid and the body of C2 and are at the greatest risk for nonunion with conservative treatment. Type III fractures were originally described to have a fracture line starting at one cortical surface of the odontoid and extending down into the body of the axis. Anderson and D'Alonzo noted that the fracture was actually a fracture of the body of C2 and not a true odontoid fracture. With the advent of CT imaging, odontoid fractures, especially type III fractures, are better appreciated anatomically. As a result of the better appreciation of the anatomic details of type III fractures, some studies have subclassified these type III fractures. Shallow type III fractures, which only extend into the cephalad portion of the body of C2, are thought to be more similar to type II fractures than to other type III fractures that have more body involvement.[15,29] Type III fractures that have more body than odontoid involvement are thought to have a better prognosis with external stabilization than type II fractures.

TREATMENT OPTIONS

Primary treatment options for acute odontoid fractures include external orthosis, odontoid screw fixation, and posterior C1-C2 arthrodesis procedures. Historically, most odontoid fractures were treated with external stabilization, and surgery was only considered after conservative treatment failed because of either gross instability of the fracture despite attempted immobilization or nonunion occurred after a period of conservative treatment.[14] As surgical methods for stabilization of odontoid fractures have improved and clinical studies have demonstrated that certain fracture characteristics are prone to nonunion with conservative treatment,
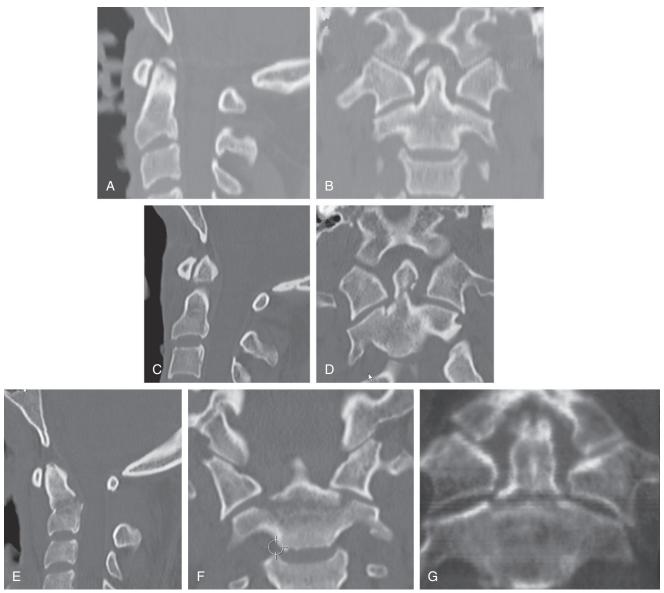
Fig. 20-4 Anderson and D'Alonzo classification is most commonly used to classify odontoid fractures. Coronal reconstruction CT images of type I *(A, B)*, type II *(C, D)*, and type III *(E, F)* fractures. *G*, "Shallow" type III fractures, as shown, are considered by many to be more similar to type II fractures than to other type III fractures with more extensive vertebral body involvement.

surgical stabilization is being considered more strongly as the primary treatment for select subgroups of odontoid fractures.

EXTERNAL IMMOBILIZATION

Options for external immobilization of these fractures include cervical collar, cervical bracing with the addition of a thoracic vest (sternal occipital mandibular immobilizer [SOMI] or Minerva braces), and halo vest immobilization.[30,31] Although sufficient clinical evidence is lacking to support one form of external immobilization over the others for the treatment of odontoid fractures, it is generally

believed that less motion (more stability) is better for bone healing, and, thus, halo vest immobilization is presumed to be better for most odontoid fractures. A cervical collar is the least cumbersome of the cervical spine orthosis options, but this comes at the cost of less support and less effectiveness in limiting motion. Studies have shown that cervical hard collars allow for more than 30 degrees of flexion-extension motion in the cervical spine.[32] Even with halo immobilization, studies have shown that 2 to 10 degrees of motion can take place at the craniocervical junction and only 75% of normal atlantoaxial motion is eliminated.[33,34]

External stabilization may be less invasive than internal stabilization but carries a substantially higher risk of nonunion, is uncomfortable for the patient, and requires longer and more stringent activity restrictions than are usually required with internal stabilization. When external immobilization is selected, odontoid fractures are typically treated for 3 to 6 months with halo immobilization and require close follow-up. We have adopted the policy of placing halos with the aid of direct lateral fluoroscopy to allow for manipulation and fracture reduction at the time of initial halo placement. This strategy tends to be more time efficient and easier on patients than serial plain films and halo adjustments. We generally follow these patients at 1, 4, 8, and 12 weeks with at least anteroposterior and lateral plain radiographs to assess alignment. In patients with less stable injuries, we often require more frequent follow-up appointments. It is important to note that some patients may have highly unstable fractures. The plain radiographs are simply a "snapshot in time," which may not reveal the full extent of subluxation. Even in halo immobilization, these unstable fractures can change alignment from minute to minute and thus the plain radiographs may not be reliable. We have observed a patient with odontoid fracture who was placed in a halo and was noted to have significant changes in alignment of the odontoid moving from an anterolisthesed to a posterolisthesed position with each respiratory cycle as observed on live fluoroscopy.

After 12 weeks of immobilization, we typically obtain a CT scan to assess for bone bridging across the fracture surface. If good bone bridging is apparent, we may discontinue the halo with possible transition to a hard cervical collar. If the bone bridging is not adequate, the patient typically is given the option of continuing with halo immobilization for up to 6 months or undergoing surgery. After 6 months, if bone healing remains inadequate, we advocate surgery. We also recommend flexion-extension studies in patients with apparent union to assess for occult nonunion and to also rule out instability from transverse ligament injury.

Halo immobilization is not a benign procedure; it is associated with pin site infections, intracranial abscess, pneumonia, and death. Lind et al.[35] found a significant decrease in vital capacity with halo vest immobilization. The elderly are especially vulnerable to complications with halo immobilization, and the authors of several studies have reported excessively high mortality in the elderly treated with halo immobilization.[21,36–38] In a review of 138 patients treated with halo vest immobilization, Majercik et al.[37] found a 20-fold increased risk of mortality (40% vs. 2%) in patients older than 65 years than in younger patients.

ODONTOID SCREW FIXATION

Another option for the acute treatment of odontoid fractures is direct odontoid screw fixation, which was first described in the Japanese literature by Nakanishi[39] in 1980. Bohler[40] later published his series of direct odontoid fixation dating back to 1968. This technique involves placing a screw anteriorly up through the body of C2 across the fracture and into the dens (Fig. 20-5). A lag screw (screw with distal threads only) is typically used to pull (or lag) the dens back into contact with the body of C2 and to provide stability to enhance the likelihood of fusion. It is important to obtain bicortical purchase at the tip of the dens because this area provides the only site for strong screw purchase within the odontoid process, and thus allows for lagging of the odontoid into approximation with the body of C2 while preventing screw pullout.

The technical challenges of odontoid screw fixation have been largely overcome with the development of guide tube systems and special tools that make this a minimally invasive technique.[41] Although odontoid screw fixation is performed without direct visualization of the surgical site, the use of biplanar fluoroscopic guidance allows precise screw placement (Fig. 20-5, A). In patients with short necks, limited neck mobility, or a large chest, the trajectory for odontoid screw fixation may be obstructed. Prior to surgery, the surgeon should always consider whether a good trajectory for drilling and placing the screw into the odontoid can be achieved. The trajectory is improved by maximizing extension of the neck, but these extension maneuvers may be limited in situations of posteriorly displaced odontoid fractures where canal compromise is a concern. This can be overcome using a special drill guide system that allows realignment of the body of C2 with the odontoid.[41]

With this technique, no bone grafting is involved; the odontoid is reapproximated and heals to the body of C2. The benefits of this procedure are that it provides immediate fixation of the fracture and, in most cases, does not require rigid postoperative immobilization. Patients may return to work earlier than when treated with halo immobilization, although we tend to restrict them to light activity for 3 to 6 months until there is evidence of bone bridging across the fracture. Another advantage of odontoid screw fixation is that it does not create a fusion between vertebrae and, thus, does not alter the biomechanics of the craniocervical junction where half of the normal rotation of the head occurs at the C1-C2 joint. Patients usually maintain their rotatory mobility because odontoid screw fixation preserves the C1-C2 articulation, but some loss of motion may occur from trauma to these joints at the time of injury.[42]

POSTERIOR C1-C2 ARTHRODESIS

The other surgical option for treatment of an odontoid fracture is a posterior atlantoaxial arthrodesis procedure. This procedure stabilizes the atlantoaxial articulation by creating a fusion across this area, but patients will lose mobility as a result. As mentioned, approximately 50% of the rotation that occurs in the cervical spine takes place at the C1-C2 articulation and this will be lost with a posterior arthrodesis procedure. In most patients this loss of mobility is well tolerated, but it should be considered as a drawback of this approach when compared with odontoid screw fixation.
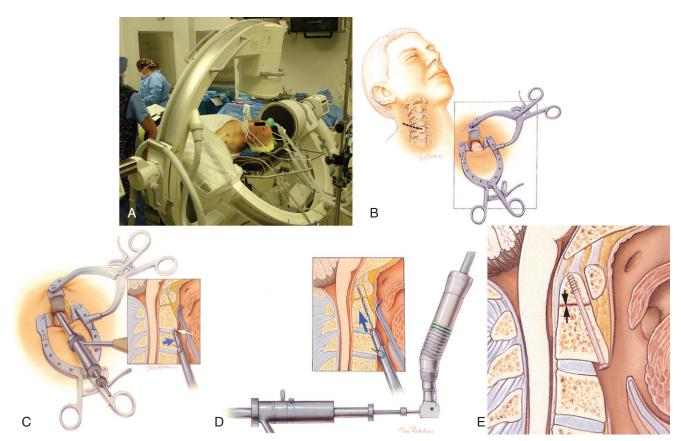
Fig. 20-5 Odontoid screw fixation can be used to treat type II and "shallow" type III odontoid fractures. *A,* The procedure is performed with biplanar fluoroscopic guidance without direct visualization of the surgical site. *B,* A Smith-Robinson or Cloward approach is used to access the prevertebral space at approximately C5-C6, and dissection and retractors are placed up to the level of C1. *C,* A K-wire is guided to the screw entry site on C2, at the anterior, inferior lip of C2. Using this K-wire, a trough is drilled into the ventral portion of the C3 vertebral body and C2-C3 disk space to provide for appropriate screw trajectory. Then, a drill guide tube with spikes is placed over the K-wire and guided to the entry site on C2. The spikes on the guide tube are impacted into the body of C3 and provide a means to manipulate the alignment of the spine to obtain a better reduction of the fracture and improved odontoid screw trajectory. *D,* Using instruments placed through the guide tube, the entire course of the screw is drilled and then tapped. Attention to drill and tap through the distal cortex of the odontoid tip is necessary because this area has the best bone for screw purchase. *E,* After drilling and tapping, a lag screw (threaded only at the distal end) is placed and used to lag the odontoid back into better approximation with the body of C2.

Numerous options exist for posterior atlantoaxial arthrodesis, including bone grafting and wiring constructs (Gallie, Brooks, or Sonntag procedures),[43] atlantoaxial transarticular screws (Fig. 20-6, *A*),[44] and C1-C2 polyaxial screws with rods (Fig. 20-6, *C*).[45] We highly recommend some form of screw fixation (atlantoaxial transarticular screws or C1-C2 polyaxial screws with rods) over wiring constructs alone because the instrumentation provides for immediate fixation, eliminating the need for external orthosis in most situations, and improves fusion rates over wiring constructs alone. The authors of some cadaveric studies[46–48] have reported no biomechanical difference between transarticular screws and polyaxial C1 and C2 screws, but these studies have not addressed the issue of translational stability, which intuitively is a concern with C1 and C2 polyaxial screws with rods in comparison with transarticular screws. In the situation of an odontoid fracture, translational instability between C1 and C2 is a major concern. Clinically, no difference has been reported in fusion rates between the two fixation techniques, but conclusive studies are lacking.

Because of our experience with atlantoaxial transarticular screw placement, we prefer this technique over C1 and C2 polyaxial screws. Atlantoaxial transarticular screws are placed by directing a screw through the posterior-inferior portion of the C2 lateral mass, across the C2 pars interarticularis and C1-C2 lateral mass articulation, and into the C1 lateral mass (Fig. 20-6, *B*). This technique, which was originally described by Grob and Magerl,[44,49] provides immediate fixation of the C1-C2 articulation and usually does not require supplemental external orthosis. The original technique involved a
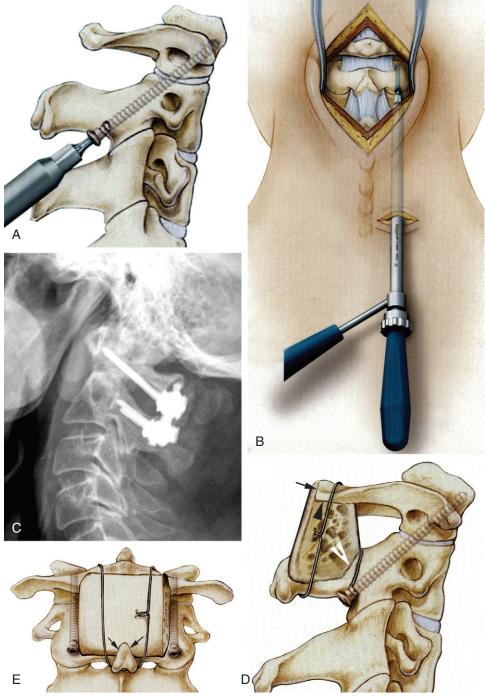
Fig. 20-6 C1-C2 posterior arthrodesis is typically performed with screw fixation and bone grafting. *A,* Atlantoaxial transarticular screw fixation involves placing a screw through the C2 pars interarticularis into the C1 lateral mass for immediate fixation of these two vertebrae. *B,* Minimally invasive techniques, using guide tubes, are commonly used to place transarticular screws. *C,* Polyaxial screw and rod fixation is another alternative to instrument C1 and C2 in posterior arthrodesis procedures (lateral radiograph shown). Bone grafting supplements the posterior instrumentation to provide for arthrodesis. *D,* Lateral illustration of our interpositional bone grafting technique. The bone graft and posterior ring of C1 is contoured to allow the graft to fit beneath (black arrow head) and behind *(thin black arrow)* the posterior arch of C1. At C2, the lamina and spinous process are decorticated and the graft is dove-tailed into the C2 lamina. *E,* Posterior view of grafting showing the notch placed within the graft to incorporate the C2 spinous process. This interpositional grafting allows loads to be passed through the graft to optimize bone healing.

long incision down into the thoracic spine to allow for angulation of instruments to obtain the proper screw trajectory; however, this was subsequently modified into a less invasive technique, involving smaller incisions, by using a guide tube system.[50]

When considering posterior atlantoaxial transarticular screws, the surgeon should closely evaluate the anatomy of the C2 pars interarticularis. The vertebral artery normally lies beneath the C2 pars and an ectatic vertebral artery or small pars interarticularis may increase the risk of injury to the vertebral artery with this procedure. We recommend obtaining fine-cut CT images (1-mm slices) and reconstructing the CT scan on a stereotactic workstation to better appreciate the anatomy of the C2 pars interarticularis for planning an appropriate trajectory for transarticular screw placement.

Even with the superior fixation of screws over wiring alone, a bone graft must be placed for bony fusion to take place. We prefer a posterior bone grafting and wiring construct with a structural graft placed behind and between the lamina of C1 and the lamina and spinous process of C2 (Fig. 20-6, *D* and *E*). Another option is bone grafting of the C1-C2 lateral mass articulation by drilling out the articulation between C1 and C2 with a high-speed drill and packing this area with bone grafting, but in our opinion this is more difficult and no more effective than a posterior bone graft secured with cables.

TREATMENT OUTCOMES AND PARADIGMS

Odontoid fractures that are not treated carry a higher risk of nonunion with the possibility of chronic neck pain and development of myelopathy because of atlantoaxial instability. The rate of odontoid nonunion with no treatment has not been adequately studied and likely depends on the individual characteristics of the fracture and the patient. The exact risk of delayed myelopathy as a result of instability from odontoid nonunion is also unknown, but many cases have been reported in the literature.[2,51–53] For these reasons, some form of treatment for odontoid fractures is recommended, with the possible exception of fractures in the severely debilitated elderly patient for whom no treatment may be a viable option.[38,54] The odontoid fracture classification system of Anderson and D'Alonzo has significantly helped stratify the best treatment options for these fractures as the different subtypes have different prognosis with conservative treatment.

TYPE I ODONTOID FRACTURES

Type I fractures are thought to be avulsion fractures of the alar ligament. Isolated type I fractures are, in general, considered stable fractures and can be treated with a cervical collar with healing reported in most cases of this rare fracture. However, Scott et al.[28] suggested that these may be unstable in some circumstances when they are part of a more extensive multiligamentous injury. Thus, evaluating these injuries for stability would seem prudent, as this fracture may be associated

with atlanto-occipital dislocation more often than an isolated fracture.[28] In the setting of atlanto-occipital dislocation, a posterior occipital-cervical arthrodesis procedure usually is warranted.

TYPE II ODONTOID FRACTURES

Type II fractures have lower fusion rates with immobilization than type I and type III odontoid fractures do, as was noted by Anderson and D'Alonzo in their original retrospective study (34% nonunion with 6 weeks of traction followed by bracing up to 6 months). Other studies have confirmed lower fusion rates for type II odontoid fractures with external immobilization and risk factors have been identified for increased risk of nonunion. The overall fusion rate for type II odontoid fractures with halo vest immobilization is approximately 70% when large published series are combined.[55,56] Small series have evaluated the cervical collar and SOMI brace for these fractures with fusion rates of 77% (26 patients) and 88% (42 patients), respectively.[30,31] Many factors, including patient age,[2,55] amount of fracture gap,[57] amount of fracture displacement,[1,2,55,57–59] posterior direction of fracture displacement,[57,58] fracture comminution,[60] and redislocation,[57] have been reported to affect the likelihood for fusion with conservative treatment, but none of these factors has sufficient clinical evidence to support guidelines in this area.[61]

Patient age is likely among the strongest factors affecting the outcome of type II odontoid fractures treated with external immobilization. The age at which patients develop a higher risk of nonunion is unclear but it is reported to be between 40 and 60 years old.[2,55] In one of the few class II studies (case-control study) in the spine literature, Lennarson et al.[55] showed a statistically significant association between age greater than 50 years and nonunion with halo vest immobilization of type II fractures. They reported a 21-fold increased risk of nonunion with halo immobilization for patients older than 50 years than for patients younger than 50 years. In addition to poor fusion rates, elderly patients also have more complications associated with halo immobilization than do younger patients.[37,38,62]

The amount and/or direction of fracture displacement has been reported by numerous authors[1,2,58,59,63] to be associated with risk of nonunion with halo immobilization, but other authors[55] have not found significant associations. In a retrospective review of 45 cases, Apuzzo et al.[2] reported 88% rate of nonunion with external immobilization in fractures with greater than 4 mm of displacement and recommended surgery as a primary treatment for these fractures. Greene et al.[1] reported a fivefold increased rate of nonunion (86% nonunion) for type II fractures with greater than 6 mm displacement when treated with external immobilization in comparison with fractures with lesser displacement. Posterior direction of displacement has also been reported as a negative prognostic factor for type II fractures being treated with halo immobilization, but other authors have found no statistically

significant association with nonunion and direction of displacement.[55,57,58] These discrepancies may occur because these measurements and observations are made on a single image. As we discussed previously, spot plain films are really "snapshots in time" and do not reflect the degree of motion nor all the directions in which displacement may occur.

Odontoid screw fixation has been reported to be successful in close to 90% of cases.[15,16,22,64] Patient age and gender do not appear to have any impact on outcomes with odontoid screw fixation.[15,16,22] In our published series of 147 patients (94% type II fractures) treated with odontoid screw fixation,[15] we found an increased risk for nonunion with anterior oblique oriented fractures and fractures more than 18 months old when treated with odontoid screw fixation. Type II fractures with an anterior oblique orientation (Fig. 20-7) had a 25% nonunion rate, compared with a 10% nonunion rate for other fractures. The overall fusion rate for fractures less than 6 months old was 88%. There was no difference in fusion rates, however, for fractures treated primarily with surgery and those treated 3 to 6 months later (usually after a failure of immobilization). However, fractures older than 18 months had a bone fusion rate of only 25% with odontoid screw fixation. For this reason, we do not recommend odontoid screw fixation for fractures older than 6 months and instead would recommend posterior atlantoaxial arthrodesis. Although some authors[15,64] have advocated two screws for additional stability because the second screw will theoretically reduce any rotatory motion between the odontoid and C2 body, clinical and biomechanical studies have not demonstrated any benefit of two screws over one odontoid screw in regard to stability and fusion rates,[15,22,65,66] thus either appears to be acceptable.

General complications associated with odontoid screw fixation, including cardiac events, pulmonary issues, urinary infections, and deep venous thrombosis, have been reported to occur in 13.5% of patients.[17] Surgery-specific complications include dysphagia, poorly positioned hardware, and hardware failure. Death, usually related to general complications in elderly patients, has been reported to occur in as many as 11% of patients.[17] Although the complications associated with odontoid screw fixation in the elderly may be relatively high, Bednar et al.[62] have reported a significant decrease in mortality in the elderly treated with early surgery and mobilization rather than conservative treatment with traction and external immobilization. In reviewing our cases of patients older than 70 years (n = 40; average age 80.5 years), we found that their fractures healed well, but the patients had a high incidence of significant dysphagia (28%) after the surgery that persisted in some patients for months.

Posterior atlantoaxial arthrodesis is also very successful in the treatment of odontoid fractures as it is in other forms of atlantoaxial instability. With modern instrumentation, fusion rates are reported to be greater than 95% for atlantoaxial arthrodesis procedures.[11,67,68] This improvement in
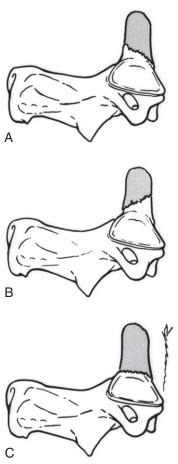

Fig. 20-7 Type II fractures can be subclassified based on the orientation of the fracture into *(A)* anterior oblique, *(B)* posterior oblique, and *(C)* horizontal. Anterior oblique fractures *(A)* are more difficult to treat and have lower fusion rates with odontoid screw fixation in comparison with posterior oblique and horizontal fractures.

success rates with posterior arthrodesis procedures comes at the cost of elimination of motion at the atlantoaxial articulation. Although no studies have addressed changes in quality of life that result from atlantoaxial fusion, most surgeons favor a motion preservation procedure, such as odontoid screw fixation, even though the success rates are slightly lower.

Considering all of these issues our recommended treatment paradigm is as diagrammed in Figure 20-8. We recommend direct screw fixation as the primary treatment of all adult odontoid fractures of less than 6 months duration but offer halo vest immobilization as an option. Patients who fail halo vest immobilization at 3 to 6 months may still undergo placement of an odontoid screw if anatomy is favorable for anterior screw fixation. Alternatively, patients who fail conservative treatment can be treated with a posterior fusion as can those who fail odontoid screw fixation or come to medical attention 6 months or more after the injury.
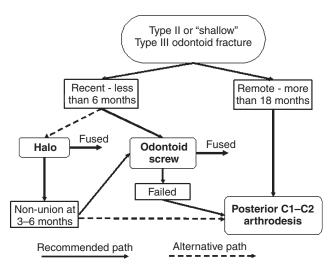
Fig. 20-8 We recommend the following treatment paradigm to treat type II and "shallow" type III fractures. In general, we prefer odontoid screw fixation for treatment of all recent (less than 6 months old) fractures. In younger patients (younger than 40 years), we consider either halo or anterior screw fixation to be a viable option, but we believe that the benefits of immediate stability with odontoid screw fixation allow patients to resume a normal lifestyle earlier in their recovery. In fractures older than 18 months, we have not had reliable success with odontoid screw fixation and therefore recommend posterior atlantoaxial arthrodesis in this scenario. For fractures between 6 and 18 months old, we do not have extensive experience to convincingly recommend one form of treatment, but we suspect that the fusion rates of odontoid screw fixation decreases with time after 6 months.

TYPE III ODONTOID FRACTURES

Type III fractures have been reported to have better fusion rates (80%–100% fusion) with external immobilization than do type II fractures.[1,20,59,63,69] It is theorized that type III fractures have a better blood supply and more surface area that account for improved fusion rates. There also may be less inherent motion because of interlocking of the irregular bone fracture surfaces. Greene et al.[1] reported bone fusion in 68 (98.6%) of 69 type III odontoid fractures treated with external immobilization. It should be noted that there may be a selection bias in many of the reported clinical series with immobilization of only minimally displaced fractures. Although sufficient data are lacking to make conclusive results, large amounts of fracture displacement (>5 mm) with type III injuries likely result in increased risk of nonunion with conservative therapy.[59] There may also be a bias toward treating the so-called shallow type III fracture with odontoid screw fixation as the primary treatment to avoid prolonged immobilization, and, therefore, it is unclear whether these fractures are included in the reported case series.

The distinction between type II and type III fractures can be difficult in some cases. Some authors consider shallow type III fractures, in which the fracture only extends into the

superior portion of the vertebral body, to be more similar to type II odontoid fractures than to other type III fractures, which have more vertebral body involvement.[14,15,29] In an effort to provide a more sound distinction between type II and type III odontoid fractures, Grauer et al.[70] proposed that fractures that extend into the C2 facets are type III fractures and shallow fractures that do not extend laterally into the facets should be considered a subtype of a type II fracture. Although this subclassification seems reasonable, at this time sound clinical evidence is not available to support any definitive management scheme for these complex fractures.

In general, we support treating shallow type III fractures like type II fractures using either halo immobilization (in young healthy patients) or surgical stabilization (preferably with odontoid screw fixation or, if necessary, with posterior atlantoaxial arthrodesis). In type III odontoid fractures with a large amount of axis body involvement, we support halo immobilization but may consider posterior atlantoaxial arthrodesis in fractures that are grossly unstable and cannot maintain in reduction despite external stabilization. Odontoid screw fixation is contraindicated in type III fractures with significant body involvement as this comminuted bone will likely not provide a firm anchor for the screw and hence the construct may fail. We have seen several cases in which the screw pulled out of the body of C2 because of unrecognized or underestimated C2 body fracture comminution.

CONCLUSION

Odontoid fractures can occur in patients of any age. In children, the fracture typically occurs at the odontoid synchondrosis and external immobilization is the primary treatment of choice because it results in excellent healing rates. In adults, odontoid fractures are much more complicated and are best treated using treatment paradigms developed from the Anderson and D'Alonzo classification system. Type II fractures are the most controversial and difficult to treat because of high rates of nonunion with conservative treatment in some groups. In younger patients, odontoid screw fixation or halo vest immobilization are both viable options. Older patients and those of any age with fractures that are hard to hold in a halo will have lower fusion rates with conservative therapy and probably should also be considered for surgical stabilization as their primary treatment. Surgical options include odontoid screw fixation and posterior atlantoaxial arthrodesis. Odontoid screw fixation is favored by most as this preserves rotatory motion at the atlantoaxial articulation, is easier to learn and perform, is a quicker operation with typically shorter hospitalization times and quicker recovery, and may be safer for the patient.

ACKNOWLEDGMENTS

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Traumatic Spondylolisthesis of the Axis/Axis Fractures

HANGMAN'S FRACTURE: HISTORICAL BACKGROUND

Although hanging was a common means of execution for hundreds of years, its results were inconsistent. Initially, the noose was tightened around the individual's neck and the individual was elevated from the ground, thus undergoing a slow and agonizing death from asphyxiation. Not surprisingly, some hangings were initially unsuccessful and required the addition of extra weights or pulling down on the victim's feet. This technique remained relatively unchanged until the late 18th century when the "long drop" was introduced. Unfortunately, decapitation was known to occur with this technique.

Because of the flaws in these approaches to execution, a method was sought that would result in death from a C2 fracture with transection of the spinal cord and not by either strangulation or decapitation. In the early methods of hanging, the knot was placed in a subaural position, creating a unique fracture through the base of the skull. In 1913, Dr. F. Wood-Jones presented a series of five prisoners from the Rangoon Central Jail who suffered instantaneous death with the knot placed in a submental position.[1] All had identical lesions in the cervical vertebrae: "… the posterior arch of the axis is snapped clean off and remains fixed to the third vertebra, while the atlas, the odontoid process, and the anterior arch of the axis remain fixed to the skull." This lesion caused instant severing of the spinal cord. This is the first known anatomical description of a "hangman's fracture."

It was not until 1954 that Grogono[2] published the first radiographs showing a fracture of the posterior arch of the axis in a patient involved in a motor vehicle accident and noted the similarity with the lesion described by Wood-Jones 40 years earlier. This description was followed by that presented by Garber[3] 10 years later of a series of eight patients who sustained "… fractures through the pedicles of the axis with displacement of the body" also after motor vehicle accidents. Garber was the first to use the term "traumatic spondylolisthesis of the axis" to describe this injury. Finally, in 1965, Schneider et al.[4] published the first definitive report exclusively on what they called "hangman's fracture." The terms *traumatic spondylolisthesis* and *hangman's fracture* have since been used interchangeably.

ANATOMY

The axis is a transitional vertebra. It bridges the cervicocranium, which is composed of the base of the skull, the atlas, the odontoid process, and the body of the axis, to the more uniform lower cervical vertebrae. One of the unique features of the axis is that the arrangement of the articulating facets is such that the superior pair is approximately one-half inch anterior to the inferior pair. In the lower cervical vertebrae, the superior and inferior facets are stacked with no displacement between them. The separation of the articulating facets in C2 divides the neural arch into three distinct segments: the pedicle, the pars interarticularis (isthmus), and the lamina.

It is important to understand that the terms *pedicle* and *pars interarticularis* refer to different parts of the C2 vertebra (Fig. 21-1).[5] The pedicle forms a posterolateral extension of the vertebral body, which connects the body to the superior articulating facet (also called the lateral mass). The pars interarticularis, or isthmus, is a narrow segment that bridges the superior facet with the inferior facet. The final segment of the neural arch is the lamina, which runs posteromedially from the inferior facet and fuses with the contralateral lamina to form the spinous process.

The early literature is confusing as to which exact anatomic structure is disrupted in a hangman's fracture. The fracture has been described as occurring in the neural arch, pedicle, lamina, ring, or body. It has also been described as an avulsion fracture of the posterior elements. The most
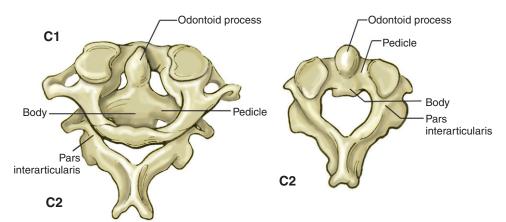
Fig. 21-1 The anatomy of C2. The pedicle and pars interarticularis are separate and distinct parts of the axis. (Reproduced with permission from Coric D, Wilson JA, Kelly DL Jr: Treatment of traumatic spondylolisthesis of the axis with nonrigid immobilization: A review of 64 cases. J Neurosurg 85:550–554, 1996.)

widely used definition is a fracture involving bilateral pars interarticularis with or without extension into surrounding structures.

EPIDEMIOLOGY AND CLINICAL PRESENTATION

Fractures of the axis account for 15% to 20% of all cervical fractures, and traumatic spondylolisthesis makes up 20% to 40% of all axis fractures.[6–9] The overwhelmingly predominant cause of hangman's fracture is motor vehicle accidents. Other causes include falls, diving accidents, and motorcycle accidents. Hyperextension with axial compression is believed to be the mechanism in most cases, and this theory is supported by the high association of facial trauma.

It is estimated that as many as 25% to 40% of patients with acute C2 fractures die at the scene of the accident.[10,11] Those that survive often have other serious injuries, including other cervical spine fractures. The incidence of other cervical spine fractures in association with traumatic spondylolisthesis ranges from 7% to 33%.[7,8,12–15] Approximately 90% of concomitant cervical fractures occur in the upper three cervical vertebrae.[12,13] Injuries to the brain (which are usually limited to concussion), chest, and extremities have also occurred with high frequency in patients with acute axis fractures.

A hangman's fracture results in a posterior decompression of the C2 vertebra, which usually results in little to no neurologic injury.[16] Reported neurologic sequelae have generally ranged from 0% to 26% of cases.[3,13,14,17–20] However, the authors of several smaller series, such as Cornish,[21] Marar,[22] and Tan and Balachandran,[23] have reported a higher incidence (36%, 73%, and 40%, respectively). In those patients who did suffer neurologic complications, clinical presentations included paresis, paresthesia, and weakness of one or more extremities, occipital pain, and Brown-Séquard lesions. Effendi et al.[19] found that of the

11% of patients who suffered a neurologic deficit in their experience, only 1.5% had a permanent deficit. Atypical hangman's fractures are those in which the fracture occurs through the posterior aspect of the vertebral body with unilateral or bilateral continuity of the posterior cortex or pedicle.[24–26] As the posterior and anterior elements separate, the spinal cord is compressed against the posterior vertebral body cortex that separates with the posterior elements (Figs. 21-2, *A* and *B*). Of the six patients reported by Starr and Eismont,[26] one suffered a complete C3 quadriplegia and another had incomplete C3 hemiplegia.

MECHANISM OF INJURY

Hangman's fractures are almost always the result of hyperextension combined with axial compression. The usual scenario is of an unrestrained motor vehicle driver hitting the windshield with his head, causing hyperextension and compression. The authors of many series have reported a high prevalence of facial trauma to support the hyperextension mechanism.[13,14,18,27] In the report by Fielding et al.,[13] 79% of patients with traumatic spondylolisthesis had face and scalp wounds, 94% of them located frontally. Fractures and soft tissue contusions in the region of the mandible and face also correlated with hyperextension.[18]

In their classic paper, Schneider et al.[4] explained how the combined forces of hyperextension and axial loading produced the hangman's fracture. With sudden hyperextension and axial loading, the weight of the skull and its contents are transmitted through the occipital condyles and atlantolateral masses bilaterally to converge at the base of the axis. The axis, serving as the transitional vertebra, then transmits these force vectors from the cervicocranium to the lower cervical vertebrae through three distinct paths: along the vertebral bodies anteriorly and through the articulating facets posterolaterally (Fig. 21-3). The lateral lines go through the weakest part of the axis, the pars interarticularis.
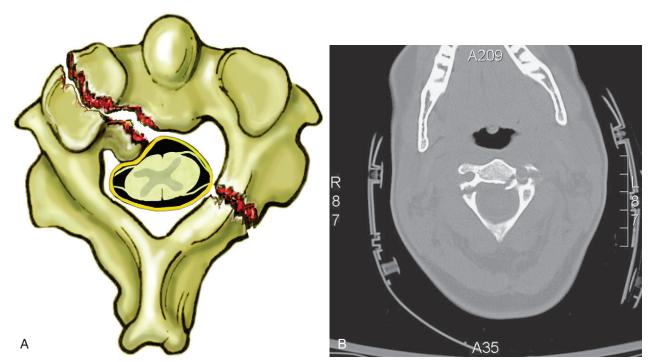
Fig. 21-2 *A,* Diagram of an atypical fracture with cord impingement. *B,* Axial CT scan of an atypical fracture in a 45-year-old woman who was involved in a motor vehicle accident. (*A,* Reproduced with permission from Starr JK, Eismont FJ: Atypical hangman's fractures. Spine 18:1954–1957, 1993.).
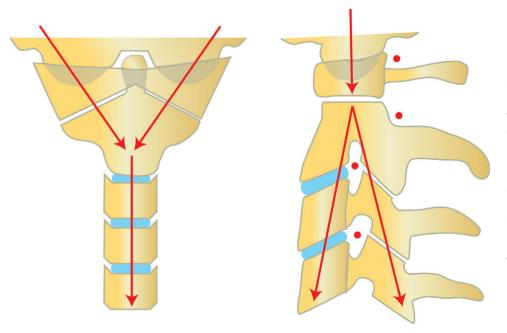

Fig. 21-3 Anterior and lateral diagrams illustrating the lines of force transmission through the atlantoaxial complex. (Reproduced with permission from Schneider RC, Crosby EC: Craniocerebral, cervicomedullary, and spinal injuries. In Kahn EA, Bassett RC, Schneider RC, Crosby EC [eds]: Correlative Neurosurgery, vol 2, 3rd ed. Springfield, IL, Charles C Thomas, 1982.)

The anterior supporting structures (anterior longitudinal ligament and intervertebral disk) are stressed with continued hyperextension. Simultaneously, the posterior bony facets are compressed until the pars fractures. With continued extension, the anterior ligament and disk will rupture with or without an accompanying body avulsion fracture of C2 or C3. The disk usually separates from the body of the third vertebra followed by rupture of the posterior longitudinal ligament. Some authors believe that it is at this stage that neurologic injury may occur.[27] With the anterior portion of C2 effectively separated from the posterior elements, the amount of anterior displacement (i.e., listhesis) is a function

of the amount of anterior ligamentous and disk damage. This displacement enlarges the spinal canal and intervertebral foramina. Thus, traumatic spondylolisthesis can be viewed as an acute posterior decompression of the axis. This mechanism has been supported by autopsy data.[28]

Flexion with axial loading can produce the same lesion.[13,17,29] This is again due to the role the axis plays as a transitional vertebra. Compression is the force vector that actually causes the fracture of the pars, with the accompanying extension or flexion component giving anterior displacement or angulation, respectively. The major difference between a flexion-compression mechanism and the hyperextension mechanism is that the posterior longitudinal ligament will be disrupted with relative sparing of the anterior supporting structures in the former. In the series reported by Francis et al.,[27] only 5 of 123 patients had physical evidence of a flexion injury, namely, wounds to the posterior vertex or occiput. All five of these patients also had compression fractures of the third cervical vertebra. Autopsy evidence supporting the flexion-compression mechanism[30] demonstrated complete disruption of the interspinous ligaments, ligamentum nuchae, facet ligaments, and posterior longitudinal ligament. The anterior longitudinal ligament, as expected, remained intact. Few authors have described flexion as a means of producing traumatic spondylolisthesis, but Levine and Edwards[17] recognized it as a valuable component in the development of their classification system, which is discussed later.

CLASSIFICATION

Three main classification systems for hangman's fracture have been described in the literature. Effendi et al.[19] based their classification scheme on the degree and type of displacement of the anterior and posterior fragments (Fig. 21-4). Type I fractures have minimal anterior displacement or angulation of the axis. Type II fractures have obvious forward listhesis or angulation in either a flexed or extended position. Type III fractures have anterior displacement with the body in a flexed position and bilateral dislocated and locked facets. In their series of 142 patients, Effendi et al.[19] observed that 65% of patients had type I fractures, 28% had type II fractures (15% with listhesis, 8% flexed, and 5% extended), and 7% had type III fractures. The authors proposed that type I fractures were the result of axial loading and hyperextension, type II occurred by hyperextension and rebound flexion, and type III by primary flexion and rebound extension.

The grading system by Levine and Edwards[17] is a modification of that devised by Effendi et al.[19] Again, the amounts of angulation and displacement were calculated to determine the grade.[17] Fractures were classified into four types (Table 21-1, Figs. 21-5, *A* to *D*). Of the 52 patients in their series, 15 had type I injuries, 32 had type II injuries (of which 3 were type IIa), and 5 had type III injuries. In this grading system, type I fractures are the result of hyperextension and axial loading

strong enough to fracture the pars but not enough to compromise the anterior and posterior longitudinal ligaments or disrupt the disk. Hence, displacement and angulation are minimal. Flexion is the primary force causing types IIa and III fractures. Type II fractures are due to a combination of initial hyperextension followed by flexion and compression. In type IIa, flexion is combined with distraction producing angulation with minimal displacement. Flexion and compression are the forces involved in type III fractures. This grading system was the first in which a combined hyperextension and flexion mechanism had been proposed. The authors proposed that flexion was the component that resulted in anterior displacement of C2. In addition, they suggested that violent flexion may cause anterior compression of the C3 body, which they found in 22 of 29 type II patients.

Francis et al.[27] developed their grading system based on cervical instability parameters as defined by White and Panjabi.[31] Fractures were classified into five grades according to displacement, angulation, and ligamentous instability. In grades I and II, displacement of less than 3.5 mm is associated with less than 11 or more than 11 degrees of angulation, respectively. In grades III and IV, the displacement is greater than 3.5 mm but the vertebral width is less than 0.5 mm and angulation is less than 11 or more than 11 degrees, respectively. Grade V is characterized by disk disruption. In their study of 123 patients, 15% of patients had grade I fractures, 7% grade II, 37% grade III, 34% grade IV, and 6% grade V.

CLINICAL MANAGEMENT

DIAGNOSTIC TESTS

The first step in the management of hangman's fractures is to obtain imaging studies. Whether patients present awake and alert with an isolated cervical injury or severely injured with polytrauma, images should be obtained to define the osseous anatomy (plain x-ray films, but preferentially computed tomography with reconstructions) and soft tissue and neurologic structures (magnetic resonance imaging [MRI]). A magnetic resonance angiography or computed tomographic angiography scan should also be performed to evaluate the vertebral arteries, as dissections and thrombosis have been reported.[32,33] We usually do not perform flexion-extension films in the acute setting.

NONOPERATIVE MANAGEMENT

The goals of treatment are to obtain adequate fusion of the fracture, to regain cervical alignment and stability, and to assist the patient to walk as soon as possible. In 2002, the American Association of Neurological Surgeons and the Congress of Neurological Surgeons jointly published Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries.[34] The authors of the guidelines reviewed the

Type I
65% stable

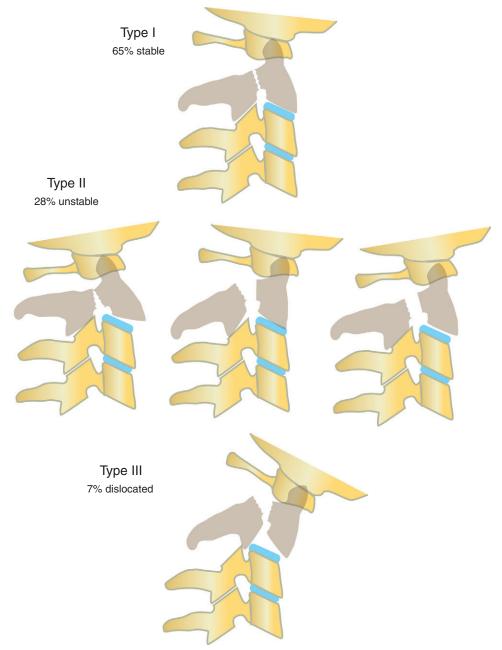Type II
28% unstable

Type III
7% dislocated

Fig. 21-4 The Effendi classification. The percentages indicate the frequency of the fracture type in their series. (Reproduced with permission and copyright of the British Editorial Society of Bone and Joint Surgery; Effendi B, Roy D, Cornish B, et al: Fractures of the ring of the axis. A classification based on the analysis of 131 cases. J Bone Joint Surg Br 63-B:319–327, 1981.)

literature on hangman's fractures and concluded that the vast majority of patients can be managed with cervical immobilization with good results.

In our opinion, Levine and Edwards types I and Ia fractures can be managed nonoperatively initially with a cervical orthosis. Type III fractures or those with disk disruption visible on MRI studies require operative fixation, although successful nonoperative management of these injuries has been reported.[35] Those patients with types II and IIa fractures are the ones in whom the management is less clear. We believe that all of these patients should be initially managed with cervical immobilization. Some authors have suggested obtaining dynamic flexion-extension films to determine which patients should be placed in a halo as opposed to a collar.[12,36] For example, Levine and Dacre[36] placed patients who had a type IIa or type II fracture with less than 5 mm of displacement on a static film but greater than 2 mm of movement on dynamic films in a halo. Surgery should be

TABLE **21-1**   Classification by Levine and Edwards[17]

| TYPE	DESCRIPTION	PROPOSED MECHANISM
I	<3 mm Displacement No angulation	Hyperextension and axial load
Ia	Oblique fracture line often extending into C2 body on one side	Hyperextension and lateral bending
II	>3 mm Displacement Significant angulation	Hyperextension and axial load with rebound hyperflexion
IIa	<3 mm Displacement Significant angulation	Hyperflexion and distraction
III	Bilateral C2-C3 facet dislocation with <3 mm displacement	Hyperflexion and distraction with rebound hyperextension
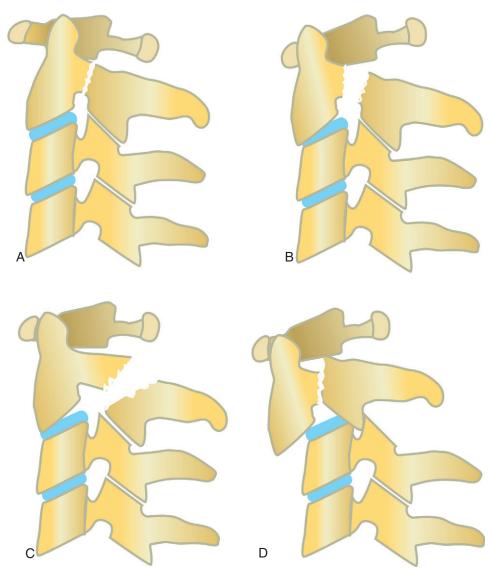
Fig. 21-5 The Levine-Edwards classification. *A,* Type I. *B,* Type II. *C,* Type IIa. *D,* Type III. (Reproduced with permission from The Journal of Bone and Joint Surgery, Inc.; Levine AM, Edwards CC: The management of traumatic spondylolisthesis of the axis. J Bone Joint Surg Am 67:217–226, 1985.)

performed in those patients in whom alignment cannot be obtained or maintained or in those patients who do not wish to wear or cannot tolerate the halo vest (e.g., elderly patients or those with significant facial trauma).

Nonoperative reduction of the fracture can be performed either with skeletal traction using tongs or with the application of the halo. In early studies, patients were placed in Gardner-Wells tongs for 3 to 6 weeks and then transitioned to an orthosis or halo.[17,19,27] If tongs are used, frequent follow-up radiographs must be obtained to confirm that reduction is maintained and avoid overdistraction because the degree of ligamentous instability is sometimes difficult to determine. Overdistraction, or "iatrogenic hanging," has been known to be fatal. Many authors advocate placing patients in halos. Vaccaro et al.[37] treated 31 patients with either type II or IIa fractures with immediate halo immobilization and found that 25 achieved fusion without reapplication of traction. Six patients did require reapplication of traction reduction and, in these patients, their initial fracture angulation was 12 degrees or greater. We and other authors avoid placing patients in a halo because it is cumbersome, disliked by patients, and associated with complications, particularly in the elderly.[38–40] Furthermore, evidence indicates that a rigid collar can work as effectively as a halo. Grady et al.[41] treated 27 patients nonoperatively. Sixteen were placed in a halo, eight in a Philadelphia collar, and three on bed rest. All patients achieved a fusion, and the authors concluded that a rigid collar could be used to treat many hangman's fractures, especially those that are minimally displaced or angulated. In a review of the literature, Li et al.[42] found that 74% of patients were treated conservatively and that all type I fractures healed with conservative treatment. Approximately 90% of Effendi type II fractures healed with conservative treatment, and 60% of Levine-Edwards type II fractures did so. However, the healing rates of Levine-Edwards types IIa and III fractures were both below 50%.

SURGICAL MANAGEMENT

The **AANS/CNS** Guidelines[34] state that surgical stabilization should be considered in cases of severe angulation of C2 on C3 (Francis grades II and IV, Levine and Edwards types II and IIa) or disruption of the C2-C3 disk space (Francis grade V, Levine and Edwards type III) or in those patients in whom alignment could not be maintained or established with external immobilization. Once the decision to pursue surgery has been made, the options include anterior or posterior approaches or both. The anterior approach via a C2-C3 diskectomy through a standard anterolateral neck exposure was the favored approach in early studies.[4,19,27,28,43] On the other hand, Seljeskog and Chou[18] argued against the anterior approach, which they believed would require "... mandibular retraction and cervical hyperextension" for adequate exposure and would aggravate an injury already caused by hyperextension. A retropharyngeal approach can provide

excellent exposure of the C2-C3 region without hyperextension of the neck.[44] One absolute indication for an anterior approach would be the presence of a traumatic disk herniation compromising the spinal cord.[45]

Posterior fusion using spinous process wiring was initially advocated for all patients with type III fractures to reduce their bilateral dislocated facets.[17,46] More recently, osteosynthesis along the pars interarticularis can be achieved by placing screws into the pars and across the fracture line (Fig. 21-6). This can be done at the C2 levels in type I or II fractures in which the C2-C3 segment is still preserved or can be coupled to lateral mass screws at C3 if the segment has been disrupted (Fig. 21-7).[36,45,47–50] The controversy between anterior and posterior approaches is exemplified in a case presented by Barros et al.[51] Of the six physicians surveyed, two stated they would perform an anterior fusion, three
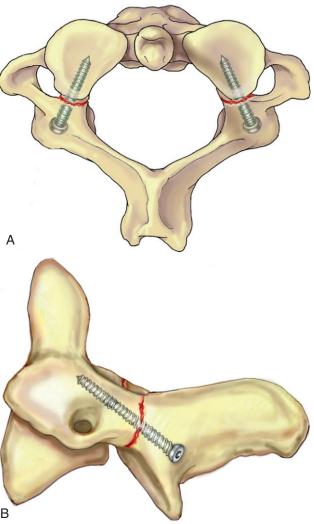

A

B

Fig. 21-6 Illustration of bilateral pars interarticularis screws. (Reproduced with permission from Bristol R, Henn JS, Dickman CA: Pars screw fixation of a hangman's fracture: Technical case report. Neurosurgery 56:E204; discussion E204, 2005.)
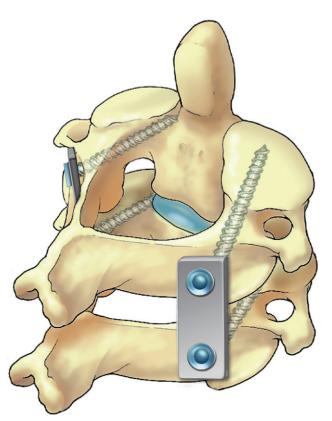
Fig. 21-7 Bilateral pars screws coupled to C3 lateral mass screws using a plate. (Reproduced with permission and copyright of the British Editorial Society of Bone and Joint Surgery; Samaha C, Lazennec JY, Laporte C, et al: Hangman's fracture: The relationship between asymmetry and instability. J Bone Joint Surg Br 82:1046–1052, 2000.)

advocated a posterior approach, and one stated he would do both. Li et al.[42] found the healing rate of type III fractures to be 39% using the posterior approach and 43% with the anterior approach and therefore advocated combined approaches for Levine-Edwards types IIa and III fractures.

PEDIATRIC HANGMAN'S FRACTURES

Although there have been numerous reports in the literature,[52–54] hangman's fractures are rare in children. In addition to other causes, in children these fractures may occur as a result of nonaccidental trauma (abuse). In some cases, they can be confused with congenital lesions, notably a persistent neurocentral synchondrosis, so-called primary spondylolysis.[55,56] The neurocentral synchondrosis, which is the cartilaginous structure that joins the body of the axis to the two posterior centers of ossification, appears at birth and ossifies between the ages of 3 and 6 years but may persist and lead to confusion.

NONODONTOID, NONHANGMAN'S FRACTURES

The third and least common type of axis fractures is called nonodontoid, nonhangman's. In the series by Greene et al.,[6] nonodontoid, nonhangman's fractures accounted for 20% of all C2 fractures. Most of them involved the vertebral body or lateral mass. Benzel et al.[57] introduced a three-tiered classification system for C2 vertebral body fractures. The type I fracture is a vertical C2 vertebral body fracture oriented in the coronal plane. The type II fracture is also a vertical fracture that is oriented in the sagittal plane. The type III fracture is a horizontal fracture of the rostral C2 body, which corresponds to the type III Anderson and D'Alonzo[58] odontoid fracture. Because this fracture type has traditionally been discussed with odontoid fractures, it is not discussed in this chapter.

German et al.[59] described nonoperative management for a series of 18 patients with vertical C2 body fractures (nonodontoid, nonhangman's fractures). All 18 patients healed with external immobilization: 13 in a Minerva jacket, 4 in a rigid cervical collar, and 1 in a halo vest because of a concomitant C1 fracture. This article suggests that a rigid cervical collar is sufficient when these fractures are minimally displaced. Greene et al.[6] also advocated nonoperative management of nonodontoid, nonhangman's fractures. Sixty of 61 patients healed with external immobilization, including halo vest, sterno-occipitomandibular immobilizer (SOMI), and Philadelphia collar. They placed patients with significant fractures of the body, pedicle, or lateral mass in a halo or SOMI for 8 to 12 weeks and those with less severe injuries in a rigid collar for 6 weeks. Other nonodontoid, nonhangman's fractures of C2 that have been reported include horizontal, Chance fractures,[60] "tear-drop" fractures,[61] and unstable coronal fractures.[62] The majority of these fractures can be managed nonoperatively.

CONCLUSION

Traumatic spondylolisthesis is a bilateral fracture through the pars interarticularis, similar to the fracture caused in judicial hangings with a submental knot. Hangman's fractures usually result from a combination of hyperextension and axial compression but may also be caused by flexion. Neurologic injury is rare with this fracture because it is essentially an acute posterior decompression of the axis with little permanent trauma to the spinal cord. Several grading schemes have been proposed, the most popular being the ones by Effendi et al. and Levine and Edwards.

Analysis of the current literature reveals that the majority of these fractures can be treated nonoperatively. Surgery is reserved for those patients whose fracture characteristics are such that conservative treatment is deemed to have a low likelihood of success (Levine and Edwards types IIa and III fractures), those with acute disk herniation causing spinal

cord compression, and those in whom conservative management has failed or who are unable to tolerate it. Surgical options include anterior, posterior, or combined approaches. Nonodontoid, nonhangman's fractures are rare, although three variations, most notably vertical-type fractures, have been reported in the literature. These can almost always be successfully treated with external orthosis.

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Distractive Flexion Cervical Spine Injuries: Unilateral/ Bilateral Facet Dislocation

INTRODUCTION

Cervical spine fractures and dislocations are of particular importance because of the close proximity to major neural and vascular structures and the high frequency of complicating concomitant injuries. Few injuries have the potential that cervical spine injuries do for catastrophic outcome and permanent disability. The vast majority of spine injuries (>70% in adults and >60% in children) involve the cervical spine, the most mobile area of the vertebral column.[1] Of the approximate 11,000 spinal cord injuries per year in the United States, 6% to 15% are the result of traumatic cervical facet dislocation, with the majority occurring at the C5-C6 level.[2–4] Forty-seven percent to 73% of cervical facet dislocation injuries occur with facet fractures and/or dislocations. Diagnosis of cervical spine injuries is a challenge, particularly in polytrauma cases where an altered level of consciousness is common.

Pediatric cervical spine injury in the United States involves less than 2% of all reported trauma, and boys are 1.5 times more likely to be injured than girls.[5,6] Some studies have reported that as many as 47% of cervical spine injuries in children occur in association with head injuries.[7–10] In contrast to adult cervical spine injuries, upper cervical spine injuries (C1-C4) in children younger than 8 years occur nearly twice as often compared with lower cervical spine injuries, with 52% of injuries located at C1-C4 and 35% of all pediatric cervical spine injuries occurring with spinal cord injury.[6]

The incidence of cervical spine injuries is likely to parallel trends in motor vehicle accidents (MVAs) and related progress in passenger safety improvement. Other important influences on incidence of cervical spine injuries are the increasing numbers of children participating in contact sports and increasing demographic trends in the aged population.[11] Cervical spine injuries comprise nearly 70% of all MVA spine injuries.[4] Most cervical spine injuries in young patients (birth to 16 years) are associated with MVAs, whereas sports-related and fall-related injuries occur more often in older age groups. High-risk sports for cervical spine injury include football (American), ice hockey, gymnastics, skiing, and diving. Cervical spine and/or spinal cord injuries represent 2% to 3% of all sports injuries.[12]

Because of the potential for ligamentous injury, intervertebral disk disruption, neurologic sequelae, vertebral artery injury, and spinal instability, diagnosis and treatment of these relatively rare but complex injuries remain a challenging and controversial area in spine care. This chapter reviews diagnostic and treatment modalities of cervical facet fractures and dislocations in the context of injury mechanisms most commonly observed with this class of injuries.

ANATOMICAL CONSIDERATIONS OF PEDIATRIC VERSUS ADULT CERVICAL SPINE

The cervical spine is divided into two regions. The upper cervical spine consists of the atlas (C1) and the axis (C2), whereas C3 through C7 compose the subaxial spine. The atlas and axis have markedly different anatomies and their most common dislocation type involves a rotatory mechanism.[13]

Pediatric spine anatomy differs from that in adults in that vertebral bodies of children are more wedge shaped. Pediatric facet joints are shallow, facet articulations, are more horizontally oriented, and ligamentous structures are relatively more dynamic and yielding than those of adults. Vertebral ossification is incomplete in the pediatric spine. The head mass and head circumference of children, relative to body size, is greater than that of adults, and therefore, biomechanical loading is greater in the pediatric cervical spine. Immature neck muscles further contribute to compromised biomechanics.[14–17]

As vertebrae of older children develop, there is a progressive loss of wedge-shaped morphology toward a more rectangular shape, and facet joints progress toward vertical alignment in the adult spine.[18–21] Other important anatomic considerations in the pediatric spine include pseudosubluxation, a normal anatomic variation at C2-C3 occurring in approximately 10% of children younger than 7 years, absence of cervical lordosis, widening of the predental space, prevertebral soft tissue widening, and intervertebral widening.[22,23]

The pediatric spine up until 8 years of age differs substantially from the adult spine with regard to structures that impart cervical spine stability, and therefore, pediatric cervical spine morphology affects trauma injury mechanisms. The anatomical differences warrant special consideration for interpretation of imaging studies, diagnosis, and management of cervical spine trauma in the pediatric patient.

INJURY MECHANISMS OF CERVICAL FACET DISLOCATIONS

Facet dislocations with or without fracture are the result of complex flexion and distraction forces and may be accompanied by an element of axial rotation.[24] The mechanisms of pathology in flexion-extension injuries and the extent to which preexisting conditions may predispose to facet dislocation in particular, continue to be debated.

Facet dislocations occur with disruption of the interspinous ligament, ligamentum flavum, and facet capsular complex. Other possible mechanisms include disruptions of the posterior longitudinal ligament (PLL) and compromise of at least a portion of the intervertebral disk.[25] The primary pathomechanism is flexion-extension force most often at the C5-C6 junction. The magnitude of the generating force, anatomical considerations, body habitus, and any preexisting deformities or degenerative conditions all contribute to a wide spectrum of injury mechanisms.

Magnetic resonance imaging (MRI) is becoming increasingly useful in characterizing soft tissue injury patterns in flexion-distraction cervical spine injuries. For example, in a study comparing unilateral and bilateral facet dislocations, unilateral facet dislocations were associated with significant disruption of the intervertebral disk, confirming findings from previous studies, and extensive soft tissue damage, with the notable exception of damage to the PLL. In the same study, bilateral dislocations were more highly correlated, compared with unilateral dislocations, to disruption of both posterior and anterior longitudinal ligaments and the facet capsule.[25] Soft tissue injury patterns may have predictive value for posttraumatic cervical spine stability. The usefulness of MRI must be considered in the context of its limitations with regard to viewer judgment and interpretation.

Elderly patients are at increased risk for cervical spine fracture and secondary neurologic decline, even after minor trauma. Hyperextension injuries in elderly patients warrant special consideration in the presence of spondylotic and/or kyphotic deformity. Subsequent infolding of the ligamentum flavum in a spinal canal already compromised by these conditions and osteophyte ingrowth may predispose such patients to spinal cord injury and neurologic compromise. Metabolic bone disease, diffuse idiopathic skeletal hyperostosis (DISH), previous spine fusion at or adjacent to the involved site, and connective tissue conditions that contribute to ligamentous laxity represent conditions in adult patients that warrant special evaluation in the presence of cervical spine injuries.

Pediatric cervical spinal anatomy differs from the adult spine in that the orientation of the facets is more horizontal and thus predisposes the pediatric cervical spine to hyperextension at the atlanto-occipital articulation. Until the pediatric spine develops intrinsic stability, ligamentous laxity may be misinterpreted as a cervical strain in the presence of a pseudosubluxation. The alignment principles of Swischuk are used to differentiate a normal vs. a pathologic subluxation.[26,27] Ligamentous laxity also contributes to the fact that the most common injury to the pediatric cervical spine occurs at the atlanto-axial junction. In the pediatric patient beyond the age of 8 years, cervical spine injuries begin to resemble those in the adult as the spine becomes more developed.[28]

TYPES OF FLEXION-DISTRACTION INJURIES

An important distinction in the classification of cervical facet dislocations is those with neurologic deficit and those without. The presence and degree of neurologic deficit correlates to the net traumatic force applied to the spine and surrounding supportive structures.[29] Braakman and Penning[30] described unilateral facet dislocations as distractive flexion injuries coupled with a rotational element. Allen and Ferguson used this concept to devise distractive-flexion injury stages, represented in Table 22-1.

Facet injuries damage surrounding ligamentous structures and may cause strains, and on occasion, complete disruptions. Bilateral facet subluxation can strain posterior ligaments and the facet joint capsule but are not considered frank dislocations. An example of a C6-C7 subluxation is shown in Figure 22-1.

TABLE **22-1**   **Classification of Distraction-Flexion Injuries**

STAGE	DESCRIPTION
I	<25% Subluxation of facets
II	Unilateral facet dislocation
III	Bilateral facet dislocation
IV	Displacement of full vertebral width

Reprinted with permission from Allen BL, Ferguson RL, Lehmann TR, O'Brien RP (eds): A mechanistic classification of closed, indirect fractures, and dislocations of the lower cervical spine. Spine 7:1–27, 1982.

Fig. 22-1 Examples of unilateral facet injury imaging studies. *A,* Model representation of a right-sided facet disloca-tion. *B,* Lateral x-ray demonstrating a C5-C6 facet fracture dislocation and a C6-C7 facet fracture subluxation. *C,* Lateral CT demonstrating rotational deformity and fracture. *D,* Axial view CT of a unilateral C5-C6 facet dislocation.

Bilateral perched facet injury involves the complete disruption of supraspinous and intraspinous ligaments as well as facet joint capsules. The ligamentum flavum and disk annulus are observed to have incomplete damage. This injury involves the alignment of the inferior tip of the superior vertebral facet and the superior tip of the inferior vertebral facet. Exaggerated kyphosis is characteristic of perching as seen on lateral radiographs.[31]

On dislocation, facets are "locked" out of anatomic position. Bilateral dislocations rarely involve rotational deformities, whereas rotation is a defining diagnostic characteristic for unilateral injury. A bilateral dislocation may cause anterolisthesis of 5 to 6 mm or 50% of the vertebral body. With this type of injury, the supraspinous and intraspinous ligaments, facet joint capsules, ligamentum flavum, and most of the disk are disrupted in their entirety.[32,33] Disk herniation may result in the extrusion of disk material into the spinal canal resulting, in neurologic deficit during or after attempted reduction.[34] Disk disruption is associated with up to 80% of bilateral facet dislocation cases.[7]

A unilateral facet dislocation often yields less anterolisthesis (<25%) of a superior segment than does a bilateral dislocation (Fig. 22-2). The contralateral facet, to some extent, still functions to maintain alignment yet also serves as a pivot point allowing anterior body axial rotation. Severe ligament damage is seen with unilateral dislocations and disk and uncinate process damage.[33] Up to 40% of unilateral cases involve concomitant disk disruption.[34]

Both unilateral and bilateral cervical facet dislocations commonly have coupled neurologic complications. Facet dislocations not involving a fracture often lead to spinal cord injury, whereas those involving a facet fracture have a lower incidence of spinal cord involvement.[10] Factors that can affect neurologic outcomes include patient age, traumatic disk herniation, time to reduction, size of neural canal, and degree of translation.

An ongoing subject of debate regarding treatment is time to reduction. Time to reduction and age were hypothesized to be factors in neurologic outcome; however, recent studies suggest that these factors do not appear to be significant determinants for recovery success.[2] Other studies suggest that a rapid spinal reduction time results in better outcomes.[35–38] Although some animal studies show that a timely decompression is advantageous for neurologic recovery,[39] no encouraging evidence exists regarding the timing of decompression procedures for human surgical candidates with neurologic deficit.[34]

CLINICAL PRESENTATION

Patients with unilateral or bilateral facet injuries may present with isolated injuries or a combination of multiple traumatic injuries. A high index of suspicion is required by the treating physician to rule out cervical spine injuries in patients with other life-threatening injuries. Although noncontiguous unstable fractures of the spine occur infrequently (3% to 8% incidence), missed diagnosis of the second lesion is common.[40]

Cervical spine instability is suspected in patients complaining of cervicothoracic pain, focal spinal tenderness, restricted neck motion, dysesthesia, and peripheral weakness. Cervical trauma patients who present without tenderness along the midline or paraspinal tissues require thorough physical and neurologic examination for angular or rotational deformities that may indicate facet compromise. A thorough understanding of the details of the inflicting trauma and the potential complications of comorbidities will enhance the success of diagnosis and treatment.

Because of the frequency of neurologic complications and deterioration with cervical spine trauma, a baseline neurologic examination at the initial stage of clinical assessment is essential. Gross neurologic status may be easily obtained in the conscious patient; however, other injuries, intoxication, or aggressive pain management may prevent a complete and accurate neurologic evaluation.

In addition to vertebral artery injury, other causes of neurologic deterioration that warrant clinical evaluation include sustained hypotension or evidence of epidural hematoma, especially in patients with ankylosing spondylitis.

Clinical evaluation of pediatric cervical spine injuries begins with a thorough assessment of posterior cervical tenderness, guarding, rigidity, or torticollis. Palpable gaps in spinous process structures may be indicative of ligamentous injury, although differentiating unossified bone and physeal cartilage from fracture may present a challenge to the practitioner unaccustomed to clinical evaluation of spine trauma in a pediatric patient. Unfamiliarity with pediatric cervical spine anatomy is a common factor predisposing to unacceptably high diagnostic error rates.[41]

DIAGNOSTIC IMAGING

Plain films are valuable in the diagnosis of traumatic injury, and include anteroposterior, lateral, and odontoid views. All seven cervical levels and the C7-T1 junction must be visualized on the lateral view.[42] A radiolucent collar is used for immobilization during imaging. If no radiographic evidence of a cervical injury exists, and the patient still complains of neck pain, additional imaging studies are warranted. Computed tomography (CT) and MRI have invaluable diagnostic capabilities. Computed tomography scans with three-dimensional reconstructions of bony anatomy render the most reliable images for determining facet injuries. The observance of unilateral facet injuries is often unnoticed on initial radiographs resulting from the impression of reduction while patients are in a supine position. A lateral radiograph may display rotational deformity through nonsuperimposable posterolateral vertebral borders or facet misalignment.[43] Examples of imaging studies of unilateral facet injuries are shown in Figure 22-3.
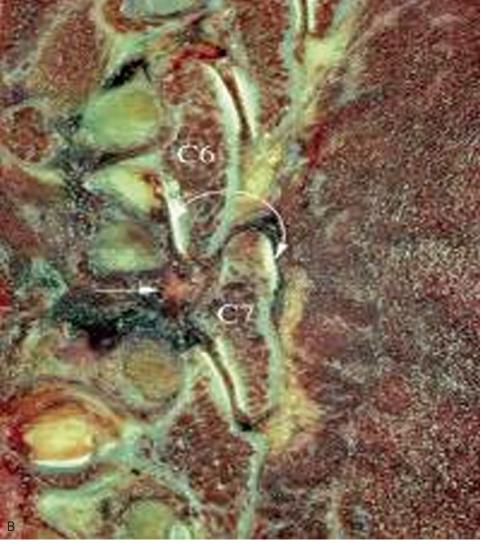
Fig. 22-2 Facet dislocation demonstrating C6 shifted anteriorly to C7. *A,* Lateral x-ray image of a C6-C7 facet dislocation. *B,* Photograph from a cadaver study demonstrating a C6-C7 facet dislocation.

Fig. 22-3. For legend see opposite page

Fig. 22-3 Imaging studies in a patient with a unilateral facet dislocation at C5 through C7, before and after surgical intervention. *A* and *B,* Preoperative lateral and anteroposterior view of a facet dislocation injury. *c* and *d,* Lateral and anteroposterior views, respectively, 2 years post fusion, demonstrating placement of anterior cervical plating.

MRI is not a reliable diagnostic tool for bone deformity, yet it is valuable for the accurate identification of soft tissue structures including ligaments, disk, and spinal cord. For patients without neurologic deficit, MRI is recommended before surgery when closed reduction has failed.[29] Reduction is indicated for all four stages of cervical distractive-flexion injuries and may proceed before an MRI examination if the patient is awake, alert, and cooperative. The patient may require a preoperative MRI if closed reduction has failed, if neurologic deterioration is present, or if a reliable examination is in question.[25] Severe flexion-distraction injuries are characterized by anterolisthesis accompanied by disk herniation. MRI is suggested for suspicion of severely herniated disks.[44,45] Reduction-induced disk retropulsion should be considered for patients with disrupted disks not yet herniated.[34] However, the lengthy time required for MRI scanning limits its efficacy for management of acute injuries.[43]

Radiologic evaluation of pediatric cervical spine injuries is similar to that in adults and includes open-mouth anterior-posterior, and cross-table lateral views. Supervised flexion and extension views are essential to assess cervical stability. In the unconscious or uncooperative patient, or spinal cord injury without radiographic abnormality (SCIWORA), MRI is desirable.

In younger pediatric patients, there should be a high index of suspicion for atlanto-occipital injuries, which can impart compromise to lower cranial nerves, greater occipital nerves, the medulla, and nearby regions of the spinal cord. Useful diagnostic evaluations in this patient group have been reviewed in detail[28] and include determination of the distance between the basion and the dens. This distance should not exceed 10 mm in children. The Powers ratio calculation (the ratio of the distance from the basion to the anterior edge of the posterior arch of the atlas, divided by the distance from the opisthion to the posterior portion of the anterior arch of the atlas) should be less than 0.9. Construction of the Wackenheim clivus line will indicate the integrity of the atlanto-occipital junction. This line is defined along the posterior clivus and should intersect or lie tangential to the odontoid.[46]

Atlanto-axial instability, also common in younger pediatric cervical spine trauma, is usually observed as an asymmetry of the lateral masses on odontoid radiologic imaging. Dynamic axial CT imaging of maximal right and left rotation may be required for a definitive diagnosis. Subaxial injuries are more common among older pediatric patients (9 to 19 years of age) as the spine is becoming more rigid, and imag-

ing studies similar to those in adults are adequate diagnostic measures. Fractures of the pedicles of C2 that occur in pediatric patients are often associated with hyperextension injuries and are difficult to distinguish from pseudosubluxation. Imaging by CT and careful assessment of the posterior laminar line are useful diagnostic indicators.[28]

CLINICAL MANAGEMENT

The goals for treatment of facet dislocation injuries are the realignment of dislocated segments and restoration of spinal stability. Ligament damage will heal with laxity and will result in compromised stability because facet joint stabilization is primarily a function of the integrity of adjacent ligamentous structures. Treatment options aimed at closed reduction include traction and manipulations. Anterior and posterior surgical options involve open-reduction maneuvers, mechanical levering, arthrodesis, and the appropriate use of instrumentation, including plating and wiring.

IMAGING STUDIES

Lateral plain films demonstrate up to 3 mm of anterolisthesis in unilateral facet dislocations. A unilateral dislocation is evident on lateral radiographs through the appearance of an anterior shift of the superior segment and lack of superimposing facets. Anteroposterior radiography will reveal a left- or right-rotated spinous process at the rotated segment level.[29] Daffner and Daffner describe a normal facet junction as having a "hamburger bun" appearance on a CT scan and describe a dislocated junction as having a "reverse hamburger bun" appearance. Figure 22-4 illustrates the phenomenon coined by Daffner.[3]

Evaluation by CT should be performed on closed reduction, and on closed reduction failure, both CT and MRI are to be performed before open approaches are considered. Those without neurologic deficit may undergo CT evaluation before the attempted closed reduction. Neurologically-intact patients should have appropriate imaging to rule out disk herniations before reduction maneuvers.

Acute cervical spine injuries and fractures in infants and children are misdiagnosed as much as 24% of the time, even in level I trauma centers.[41] The occiput to C2 presents the most challenging site for diagnosis, and failure to recognize elements of pediatric anatomy is the primary reason for missed diagnosis by radiographic evaluation. SCIWORA occurs in children younger than 10 years.
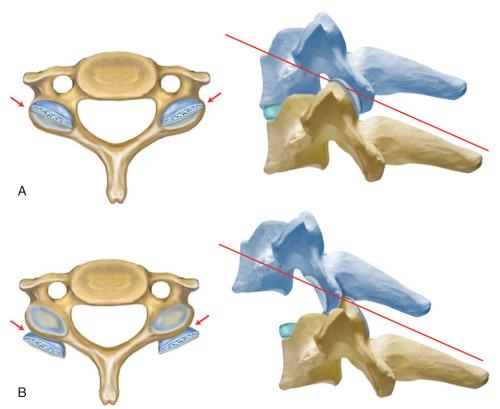
Fig. 22-4 Diagrammatic representations of dislocated facet segments. The two, mirror-image, convex aspects of a normal facet joint resemble a hamburger bun *(A)*. The reverse hamburger bun refers to a dislocated joint, with the normal convex surfaces of the joint appearing side by side *(B)*. From Daffner SD, Daffner RH: Computed tomography diagnosis of facet dislocations: the "hamburger bun" and "reverse hamburger bun" signs. Journal of Emerging Medicine 23(4):387-94, 2002, reprinted with permission.

NONOPERATIVE APPROACHES

Proper management of facet dislocation injuries involves reduction and stabilization, with closed techniques demonstrating good success in the moderately-injured patient. In one recent study, closed-traction approaches were used successfully in as many as 88% patients, whereas the remaining 11.2% required open management.[2]

Perched facets should be treated with closed skeletal traction. Reduction with Gardner-Wells tongs may be attempted with an initial weight of 5 pounds and subsequent 5-pound increments until adequate axial lengthening and acceptable disk positioning is clear. Sequential weight increases should be monitored with neurologic examination and radiographic imaging of the distance between facets to avoid overdistraction. Fluoroscopic guidance or serial plain radiographs should accompany reduction maneuvers.[47] A weight of 50 pounds is generally enough for reduction of cervical facet dislocations.[29] However, there is no consensus in the literature regarding a safe weight limit for closed reduction maneuvers.[48]

Initial management of the pediatric patient with a cervical spine injury should begin with appropriate transport. Infants and children younger than 10 years require the cervical spine to be in a neutral position with a modified spine board, allowing for a larger head-to-torso ratio. In children younger than 12 years, halo traction with modified pin placement is preferable to cervical traction with Crutchfield tongs, which have been associated with dural complications.[22] Halo placement may benefit from CT evaluation to determine skull thickness and assist in the avoidance of suture lines, the temporal region, and frontal sinus. Halo placement is contraindicated in children younger than 2 years, and for children older than 8 years, a standard adult four-pin configuration is adequate. Halo placement in children within the intermediate age group (2 to 8 years old) should use a 6- to 12-pin configuration. Pin site infections from halo use are common in children, occurring in as many as 40% of placements.[49,50] For neurologically intact children with stable cervical spine injuries, a rigid orthosis or Minerva brace for 8 to 12 weeks is usually adequate treatment.

INDICATIONS AND CONTRAINDICATIONS FOR SURGERY

Surgical versus nonsurgical management of cervical facet dislocation injuries requires individualized management decisions. However, common criteria for surgical intervention include persistent spinal instability following closed-reduction attempts and persistent or evolving neurologic deficit. In the

presence of significant disk herniation, anterior decompression is recommended.[32]

Reduction of a bilateral facet dislocation and preparation for surgery may occur up to 6 weeks following trauma. If traction fails to reduce the injury, a combined anterior/posterior fusion approach is considered. Within the operating room, patient positioning is recommended on a table with a supportive faceplate, prone-to-supine transition capabilities, and, often, the use of Gardner-Wells tongs. Head positioning should be neutral (Fig. 22-5).

If previous closed reduction has been successful, the anatomic alignment must be preserved in the operating room. Longitudinal traction of 20 to 25 pounds should sustain alignment. If the patient is flipped prone, a lateral radiograph should be taken to assess neck orientation. Spinal cord monitoring may involve the concurrent use of free-run electromyography, somatosensory evoked potentials, and transcranial motor evoked potentials during open reduction.[13]

Indications for surgery in pediatric patients are the same as those for adults, with similar treatment goals for spinal cord decompression and spinal alignment. Early surgical treatment is indicated for unstable fractures and injuries associated with spinal cord compromise. Reports of surgical treatments of unstable cervical spine injuries in pediatric patients commonly involve posterior fusion with posterior wiring. In general, the use of spinal implants in the pediatric spine is controversial because of effects on skeletal maturation.

OPERATIVE TECHNIQUES

The operative approach for cervical facet dislocations is fundamentally based on diskal integrity. If there is an MRI-identified disk herniation, an anterior decompression and concomitant reduction procedure is recommended over closed reduction because disk material may shift during manipulation and negatively impact neurologic status. Intra-

Fig. 22-5 Patient positioning for cervical spine surgery using a traction frame. Within the operating room, the patient is positioned on a traction frame with a supportive faceplate and prone-to-supine transition capabilities. The head position is neutral.

canal disk material may be dissected and removed before the spinal reduction is performed. Occasionally, the ventrocaudal edge of the anterolisthesed vertebral body may obscure the direct visualization of the disk material. In this situation, partial corpectomy may be necessary to achieve an adequate decompression.

To minimize complication, risk on reduction, the posterior longitudinal ligament, and all intracanalicular disk material should be resected. A bilateral reduction can also be performed ventrally using vertebral body posts or distraction pins for manual levering. The pins are inserted from 10 to 20 degrees relative to each other in the sagittal plane. Bringing the pins close together will create a slight kyphosis. The disengagement of the dislocated facets will be completed after a manual posterior force is applied to the rostral body (Fig. 22-6).

The reduction of a unilateral facet dislocation is relieved in a similar manner but with initial placement of the pins with a coronal separation of 15 degrees. When a slight kyphosis is manipulated, and when the pins are drawn together laterally, again, a manual posterior force is applied to the rostral body and facet joint reengagement occurs (Fig. 22-7). An intraoperative radiograph will assess the degree of reduction before pin removal. If, after radiographic analysis, the spine has not reached anatomic alignment, then the technique described is repeated. Once an adequate decompression of the spinal cord has been achieved and alignment has been restored, an interbody allograft, autograft, or mechanical device and an anterior plate may be used to stabilize the spine. For cases without severe disk herniation, a closed reduction and posterior fusion with instrumentation is recommended. If closed reduction is unsuccessful, open reduction and internal fixation and arthrodesis may be the best surgical alternative.

Traditional intraoperative imaging methods may fail in the cervical spine trauma setting in which anatomical landmarks are often disrupted. Surgical navigation technology including automated neurophysiologic monitoring, frameless stereotaxy, and three-dimensional CT imaging, all have the potential to improve the accuracy of spinal implant placement and osseous decompression.

In the pediatric surgical candidate, a posterior approach is commonly taken to treat craniocervical and subaxial instability. In the upper cervical regions of the pediatric spine, posterior instrumentation options include wiring of C1-C2, atlanto-axial fixation and occipitocervical plating. Instrumentation alternatives for the lower cervical spine include spinous process wiring, plate/rod constructs, and hook fixation. Anterior approaches are reserved for anterior compression of the spinal cord, severely compromised disk integrity, or the need for anterior column support.

COMPLICATION MANAGEMENT

Open posterior reduction often involves a modified laminar spreader with notched tips to hook the laminar lips.[51] This technique is contraindicated in patients with laminar
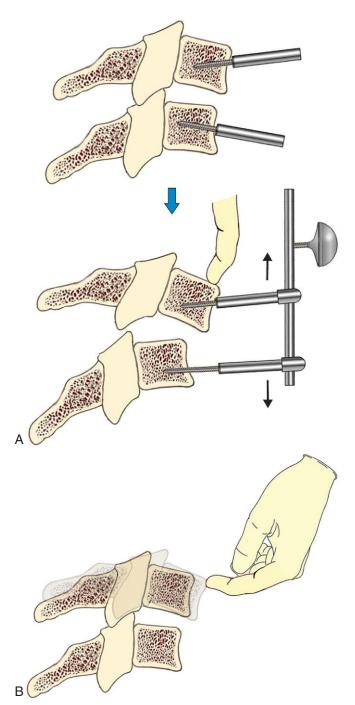
A

B

Fig. 22-6 Representation of bilateral ventral reduction vertebral body posts, inserted from 10 to 20 degrees from one another in the sagittal plane. A slight kyphosis is created, followed by a manual posterior force to the rostral body. (From Ordonez BJ, M.D., Benzel EC, M.D., Naderi S, M.D., Weller SJ, M.D: Cervical facet dislocations: techniques for ventral reduction and stablization. Journal of Neurosurgery: Spine 92(1), 2000, reprinted with permission.)

fractures in which there is a risk of separation of the lamina from the anterior body.[13]

Vertebral artery injury occurs with 11% frequency in cervical spine trauma. Clinical findings are not highly sensitive to
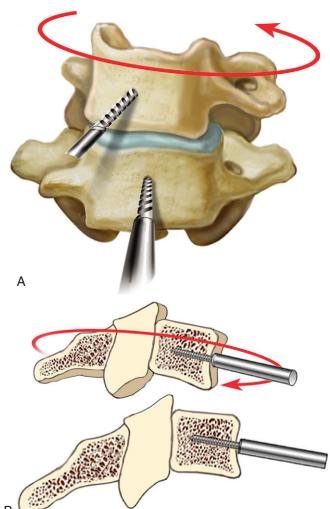

A

B

Fig. 22-7 Representation of an anterior manipulation on Caspar pins for the reduction of a unilateral facet dislocation. Initial placement of distraction pins involves 15 degrees of coronal separation. A slight kyphosis is manipulated, and pins are drawn together laterally, a manual posterior force is applied to the rostral body. (From Ordonez BJ, M.D., Benzel EC, M.D., Naderi S, M.D., Weller SJ, M.D: Cervical facet dislocations: techniques for ventral reduction and stablization. Journal of Neurosurgery: Spine 92(1), 2000, reprinted with permission.)

vertebral artery compromise in cervical spine injuries, and patients with dislocation or fractures may require special diagnostic testing.[52]

Children are at risk for posttraumatic or progressive spinal deformities following cervical spine injuries. Cervical kyphosis following multiple laminectomy for decompression is rare in the cervical trauma setting. However, kyphosis may develop in the absence of facet injury manifesting as wedging deformities of the vertebral elements and resulting in functional instability.

Paralytic spinal deformities occurring in pediatric spinal cord injury patients is a phenomenon unique to the pediatric population, and occurs in 40% to more than 90% of pediatric
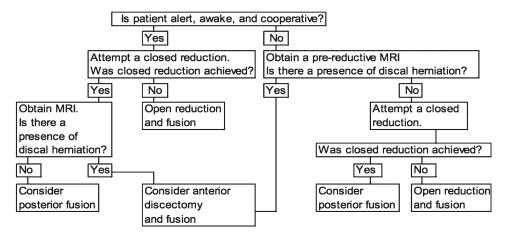
Fig. 22-8 Management algorithm for early clinical decision making in the treatment of cervical facet dislocation injuries. Early decision-making strategies are affected by the patient presentation, the success of closed-reduction maneuvers, and the presence of disk herniation.

spinal cord injuries.[53] The degree of deformity is highly correlated to the timing of injury in relation to adolescent development, with the highest risk present if the injury occurs before the adolescent growth spurt.

Careful attention should be given to surgical dissection in the pediatric spine, as iatrogenic fusion is a well-known complication and pediatric anatomy may not accommodate instrumentation as predictably as in the adult.

SURGICAL OUTCOME STUDIES

Class I evidence directly comparing surgical techniques for management of cervical facet dislocations is lacking in the current literature. Biomechanical studies conducted in cadaveric models have investigated spinal stability following various surgical techniques. In biomechanical studies modeling bilateral facet dislocations, anterior cervical diskectomy and fusion with posterior transpedicular screw/rod fixation demonstrated the most effective stabilization with regard to range of motion and neutral zone loading, followed by fixation using an anterior locking plate plus posterior wiring. The least biomechanically sound fixation tested was fixation with anterior locking plate only. However, all fixation strategies tested demonstrated higher biomechanical stability than the intact spine in most loading tests.[32]

Lateral mass screw and rod or plate constructs have been introduced in the context of minimally invasive approaches for cervical spine injuries, including facet dislocations. Preservation of soft tissue integrity contributing to the posterior tension band of the cervical spine and less postoperative wound pain are among the advantages to this approach; however, careful patient selection is imperative.[54,55]

Surgical outcomes in cervical spine trauma are likely to be increasingly improved with the use of new grafting technologies including the use of graft extenders and bone morphogenic proteins (BMPs). The use of these technologies has not been rigorously evaluated in the context of cervical trauma, and the costs may be prohibitive. Therefore, use should be considered on an individual basis or as part of a well-designed clinical trial protocol.[56]

CONCLUSION

Severe hyperflexion-distraction injuries in the cervical spine have the potential to result in damage to intervertebral disks and soft tissues including major stabilizing ligamentous structures. Disk herniation poses a high risk for neurologic compromise. Facet dislocations generally present with exaggerated kyphosis and instability; however, an appreciation of developmental anatomy in the pediatric patient is required for effective diagnosis. Management strategies in both pediatric and adult patients consist of closed reductions, followed by the appropriate surgical approaches in the presence of instability or neurologic compromise. A management algorithm for early clinical decision making is presented in Figure 22-8. Surgical techniques using various fixation strategies demonstrate a high level of biomechanical stability. Minimally invasive techniques and biologic enhancers of bone fusion represent two areas of potentially significant improvement in the treatment of facet dislocation and fracture injuries.

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Cervical Burst Fractures: Compression Flexion/ Compression Extension Injuries

INTRODUCTION

BACKGROUND AND EPIDEMIOLOGY

The Egyptians detailed the first known recordings of cervical spine fracture and spinal cord injury (SCI) more than 5000 years ago. Most injuries then, as now, were the result of blunt trauma to the head or neck. Treatment was limited because interventions did not benefit the patient, especially when there was a concomitant spinal cord injury. Little could be done for the bony component of a spinal injury because there were no means to stabilize the vertebrae. Therefore, expectant management was the rule of the day.[1] Fortunately, determined physicians sought ways to advance the treatment of these injuries. As medical knowledge progressed, recognition of the relative contributions of bone and soft tissue structures to the stability of the spine were identified and a greater understanding of the location and function of neural elements was determined. Claudius Galenus of Pergamum (131–201 AD), better known as Galen, made significant contributions with his public vivisection of the neural elements of live pigs and other animals. Around AD 600 Paul of Aegina performed the first recorded laminectomy on an injured human patient, indicating awareness by physicians of the time of the importance of the neural elements and the beneficial effects of decompression, including possible return of some neurologic function. Our current treatment concepts of spinal stabilization and neural element decompression for cervical trauma are an outgrowth of our predecessor's

investigation and experimentation. Modern advances in technique and technology offer improving outcomes for patients with these severe injuries.

The exact incidence of cervical burst fracture in the United States is difficult to determine. A recent multicenter study of 33,922 blunt trauma admissions demonstrated cervical spine injuries in 2.4%.[2] More than 50,000 spinal fractures occur each year, and by some estimates three quarters of these are cervical spine injuries. Around 25% of all spinal fractures involve a SCI.[3] No epidemiologic study exists pertaining to the specific incidence of cervical burst fractures or associated SCI. The morphologic pattern of bone injury in cervical burst fractures involves frequent incursion by bony fragments into the space available for the cord (SAC). This makes SCI an expected finding. The risk of neurologic injury in this type of injury is superseded only by bilateral facet dislocations.[4] Most patients with cervical burst fracture and neurologic injury are young Caucasian males involved in motor vehicle accidents.[5]

Patients with a cervical burst fracture usually present in one of two ways. The first type of patient is one with an isolated cervical injury. This patient's injuries usually result from lower energy trauma, such as diving into a shallow pool. Often they are conscious and can describe the accident and subsequent neurologic injury, if present. The second type of patient is the polytrauma patient who may have significant distracting injuries and an altered level of consciousness secondary to intoxication, pharmacologic intervention on the part of the trauma team, or head injury. High-energy motor vehicle accidents are the most frequent cause of injury in this group. There is a risk of the surgical team missing cervical injuries in this setting unless a thorough evaluation of the patient is performed according to *ATLS* guidelines. This includes appropriate lateral cervical radiographs or equivalent computed tomography (CT) scans. Often there is no obvious deformity present on physical examination. Bohlman[6] found as many as one third of cervical injuries may be missed at presentation, with diagnosis delayed up to a year from injury. All patients with a suspected cervical injury or altered level of consciousness should have a well-fitted cervical orthosis placed.

The importance of a thorough physical examination in the setting of cervical burst fracture cannot be overstated. It is important the spine surgeon remember other physicians on the treatment team may not be skilled at the neurologic examination. The identification of SCI is paramount. Prognosis of injury and timing of surgical intervention are determined primarily by the physical examination and categorization of neurologic deficit.[4,5]

It is the responsibility of the spine surgeon to ensure appropriate plain films and advanced imaging studies are obtained. If the quality of imaging is not adequate, new images must be obtained. Thin-cut (3 mm) CT scans with sagittal and coronal reconstructions are the most helpful imaging modality for surgical planning as they afford a three-dimensional analysis of the injury and assist in surgical planning (Fig. 23-1). After the plain lateral cervical spine film in the trauma bay, CT should be the next study obtained because other imaging modalities, such as magnetic resonance imaging (MRI) require a significant amount of time, may not be necessary with a good physical examination, and may delay surgical treatment. MRI is not as important as CT when a good physical examination can be obtained but may be very useful to elucidate SCI (see Fig. 23-1) in the setting of altered consciousness. It also helps with assessment of the ligamentous supportive structures of the spine.

CLINICAL MANAGEMENT

CLASSIFICATION OF INJURY

Cervical burst fractures are the result of compressive forces on the vertebra. Several different classification systems have been developed in an attempt to assign anatomic patterns of injury to mechanism of injury. The most often used of these is the classification developed by Allen et al.,[7] which is quite inclusive. There are many different types of injuries in the Allen classification that may appropriately be called burst fractures (Table 23-1). The problem with mechanistic classifications is they are complicated, and anatomic injury patterns often do not adhere to the mechanism into which they should fit.

The descriptive classification of Denis[8] is useful both as a tool for communication, because of its simplicity, and as a determinant of treatment because it connotes stability. Denis describes the spine in terms of three columns: the anterior, middle, and posterior. Burst fractures are defined as injuries that include damage to structures of the anterior and middle columns and may include injury to the posterior column as well. White et al.[9] defined clinical instability as "the loss of the spine under physiologic loads to maintain relationships between vertebra in such a way that there is neither damage nor subsequent irritation to the spinal cord or nerve roots and, in addition, no development of deformity." Burst fractures in the cervical spine are by this definition, unstable injuries.

The favored approach to treatment of cervical burst fractures involves two distinct goals. The first, and least controversial, is restoration of stability and anatomic position of the spine. The second is decompression of the neurologic elements of the spine. After radiographic images have been obtained, an assessment of the anatomic location of fracture planes and of the bone and soft tissue structures violating the SAC should be made. It is important to recognize the amount of SAC violation present on radiographs does not indicate the amount of occlusion at the moment of injury.[10] Likewise anatomic position of vertebral elements on evaluation imaging studies does not connotate stability. The position of the head can significantly alter SAC violation, with a slightly flexed position offering reduction and a greater SAC.[11] Although greater SAC occlusion increases the probability of neurologic injury, there is no direct correlation with presence or severity of neurologic injury and canal violation on presentation. Rather it is the force of the initial impact on the neurologic elements and persistence of compressive pathology and edema that seem to ultimately determine the severity of SCI.[12,13]

Given the instability demonstrated by cervical burst fractures, intervention on the part of the treating spinal surgeon is mandatory. Several treatment options do exist however, and the surgeon may choose to proceed either operatively or nonoperatively. The halo vest orthosis is the mainstay of nonoperative treatment, although historically, long-term hospitalization and cranial traction was also used. Koivikko et al.[14] compared the results of nonoperative and anterior operative treatment of cervical burst fractures in a retrospective study of 69 patients. Thirty-four patients were treated nonoperatively and 35 operatively. They found surgical treatment to be superior to nonoperative treatment in terms of neurologic recovery ($p = .027$), spinal canal decompression ($p = .0006$), and resolution of kyphotic deformity ($p = .00003$). They also noted a trend toward increasing kyphotic deformity in the nonoperative treated group not present in the surgically treated group. Similar rates of union were observed in the two groups. Pharmacologic intervention with intravenous methylprednisolone did not seem to affect the outcome positively or negatively. Interestingly, the complication rate was similar in the two groups.

Although the previous study suggests better neurologic functional recovery in patients treated operatively, significant debate exists as to the timing of surgical intervention and its effect on neurologic outcome. Pollard and Apple[5] performed a large retrospective study to evaluate factors associated with improved neurologic outcomes in patients with incomplete SCI. They evaluated 412 patients with traumatic, incomplete, cervical spinal cord injuries with an average follow-up period of 24 months. Neurologic recovery was not significantly related to any of the following factors: gender, race, type of fracture, mechanism of injury, high-dose methylprednisolone (*NACIS* II protocol), early definitive surgery, early anterior decompression for burst fractures or disk herniations (defined as < 24 hours from injury), or
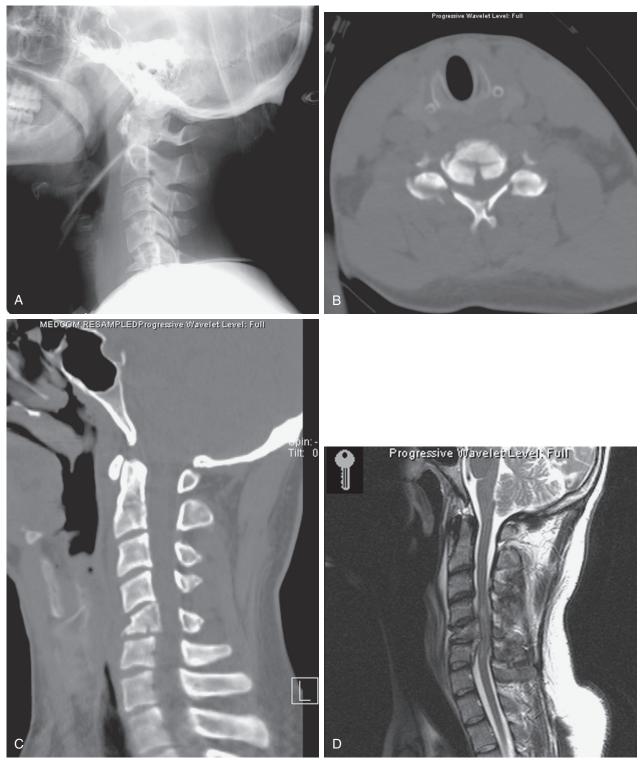
Fig. 23-1 A 26-year-old man with fall into shallow pool and complete SCI. *A,* Trauma bay cross-table lateral demonstrating fracture of C5 vertebra. *B,* Thin-cut axial CT image demonstrating three-column injury with canal compromise. *C,* Sagittal CT image demonstrating kyphosis and fragment retropulsion. *D,* Axial T2 MRI image demonstrating canal compromise and cord signal changes from SCI. *E,* Intraoperative photograph demonstrating lateral mass screw fixation and posterior decompression of spinal cord by laminectomy. *F,* Intraoperative photograph demonstrating anterior plate fixation. *G,* Postoperative AP radiograph demonstrating anterior plating with posterior lateral mass screw fixation and PEEK structural cage with autograft. *H,* Postoperative lateral radiograph with restoration of sagittal contour of cervical spine.
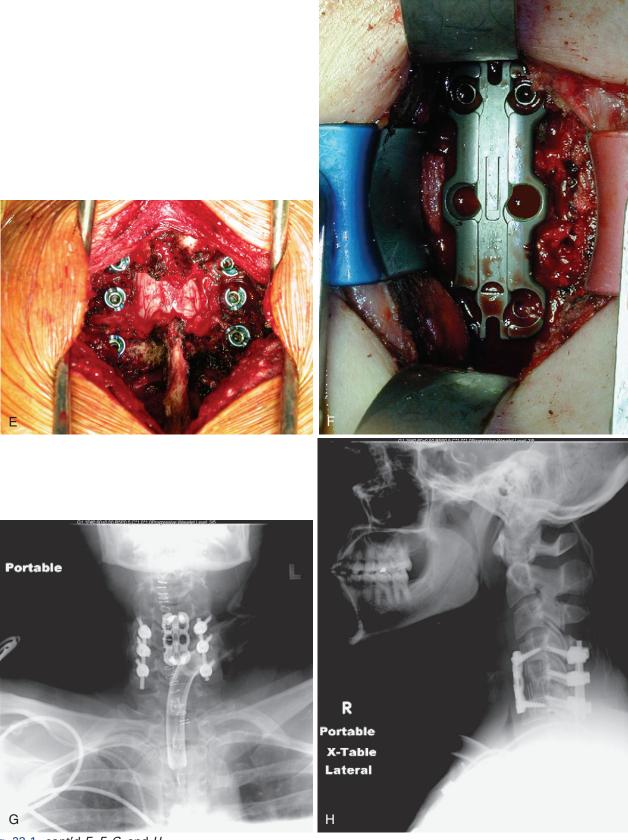
Fig. 23-1, cont'd *E, F, G,* and *H*

TABLE 23-1   Allen and Ferguson Injuries That May Constitute Burst Fractures[7]

TYPE/STAGE	CHARACTERISTICS
COMPRESSION FLEXION (CF)	
CFS4	Beak fracture with decreased body height plus <3 mm displacement at the fracture site
CFS5	As above with displacement of the vertebral body into the canal, may have complete posterior ligamentous disruption
VERTICAL COMPRESSION (VC)	
VCS2	Fracture of both end plates with minimal displacement
VCS3	VCS2 with fragmentation and displacement of the vertebral body
COMPRESSION EXTENSION (CE)	
CES4	Bilateral arch fracture with partial displacement of the body
CES5	Bilateral arch fracture with complete vertebral body displacement

decompression of stenotic canals without fractures. The significant factors for improved neurologic outcomes were young patient age ($p = .002$) and those with either a central cord or Brown-Séquard syndrome ($p = .019$).

The finding of no improvement with early decompression is at odds with experimental research in the laboratory which has demonstrated significant improvement in animal models with early decompression.[13] Some of this difference might be resolved with clinical studies which define early intervention of less than 12, 8, or 6 hours, but this body of evidence does not currently exist in the clinical arena. The body of evidence we rely on in the clinical realm is weakened by its retrospective nature.

Debate also exists as to the most appropriate type of surgical procedure, a stand-alone anterior decompression procedure with plating, or a combined anterior and posterior procedure. There is little debate among the researchers that like to break bone that a combined anterior and posterior procedure affords greater immediate stability compared with either anterior- or posterior-based approaches alone in cadaver studies.[15–18]

Every surgeon must individualize the care of each injured patient. We use philosophical and scientific criteria to determine an operative strategy for each injury. In general, we believe the best opportunity for neurologic recovery is early surgery with neurologic decompression. In addition, early rigid stabilization with combined anterior and posterior fixation allows for vigorous early mobilization and rehabilitation decreasing postoperative complications and giving the surgeon and patient confidence in a smooth course toward fusion and recovery.

OPERATIVE PROCEDURE AND TECHNIQUE

ANATOMY

Although a complete discussion of cervical spine anatomy is beyond the scope of this chapter, and is covered elsewhere, several salient points deserve attention. The vertebral bodies in the subaxial spine (C3-C7) have an anterior inferior slope of the superior endplate. The facet joints have the opposite orientation, preventing anterior-posterior translation of the vertebra on one another. The superior endplate is also concave rising laterally to the uncinate processes and uncovertebral joints. These are important surgical landmarks because they can be identified during the anterior approach to the cervical spine with the tip of the uncinate process defining the most lateral extent of the vertebral body. This concavity mating with the convexity of the inferior endplate prevents lateral translation of the vertebra on one another. The lateral mass of C7 may be identified intraoperatively by its smaller size. The nerve root and neuroforamina can be identified directly anterior to the superior articular process of the inferior vertebrae at the level of the facet joint.

The vertebral artery is the first branch off of the subclavian artery. It enters a foramen transversarium of C7 in 10% of cadaveric specimens.[19] The most medial extent of the foramen transversarium lies near to the posterior half of the vertebral body, with the vertebral artery exiting and running anterior to the nerve root in the neuroforamina before entering the next foramen transversarium. Often, the pedicle of C7 and occasionally C6 is large enough to accept screw instrumentation.

Palpable bony landmarks for surgical orientation include the large and palpable spinous processes of C7 (vertebra prominens), and C2 posteriorly. Anteriorly, Chassaignac's tubercle can be palpated lateral to the midline and anterior to the C6 foramen transversarium. The cricoid membrane at is C5-C6 interspace, the prominence of the thyroid cartilage overlays the C4-C5 interspace, the hyoid bone is at the C3 level, and the angle of the mandible is at the C2 level.

OPERATING ROOM SETUP

Every surgical procedure proceeds in terms of ordered phases with set and definable goals that must be accomplished before moving to the next step of the procedure. Ordinarily we

prefer an anterior and posterior approach for these complex and often highly unstable injuries. Surgical treatment of cervical burst fractures proceeds in the following order: Initiation of anesthesia, patient positioning for anterior cervical approach, sterile prep and draping, anterior cervical approach, vertebral corpectomy and neurologic decompression, structural graft selection and endplate preparation, placement of structural graft, plate fixation, closure of anterior wound, re-positioning for posterior cervical approach, sterile prep and draping, posterior approach, posterior instrumentation, decortication and bone grafting, and posterior wound closure.

Careful attention to preoperative planning using high quality radiographic studies, operating room (OR) setup, and patient positioning affords the greatest opportunity for success in the surgical treatment of cervical burst fractures. Although technically challenging, the individual technical elements of treatment should be familiar to most surgeons facile in the treatment of disorders of the cervical spine. It is important that the surgeon be present for the intubation and positioning of the patient to avoid iatrogenic exacerbation of SCI. Communication with the anesthesiology team regarding the instability of the cervical spine is mandatory. Fiberoptic intubation may be preferable to conventional intubation as less manipulation of the cervical spine is necessary. It is recommended that hypotension be avoided preoperatively, perioperatively, and postoperatively with a target MAP of 80 to 85 mmHg to be maintained pharmacologically as necessary.[20,21] Fractures tend to have increased blood loss compared with similar procedures on noninjured vertebral segments and placement of an arterial line for accurate intraoperative pressure monitoring and to allow for arterial blood gas assessment is recommended. Patients with cervical spinal cord injuries often require lengthy surgical intensive care unit hospitalizations and intravenous (IV) fluid and pressor support. It is wise to place a triple-lumen central venous line in the operatorium for postoperative management. The patient should also have two large-bore IV lines placed for purposes of fluid and blood transfusion.

PATIENT POSITIONING

For the anterior approach the patient is positioned supine on the surgical table. A "time out" is performed confirming patient identification, procedure, availability of instrumentation, graft extenders, and blood components. If a combined anterior and posterior procedure has been selected, the patient is fitted with Mayfield tongs in a sterile fashion. A rolled sheet, towel, or IV bag bump is placed centrally beneath the patient between the shoulder blades and inferior to level of surgery so as not to interfere with intraoperative fluoroscopy. The surgeon must work with the anesthesia team to ensure the breathing tube is in a position where it will not be contacted by the surgical team after draping and during the surgical procedure. The head

is placed on a foam doughnut and 5 pounds of traction is applied through the tongs. The shoulders are depressed and taped to the foot of the bed. Careful padding of all bony protuberances and neurovascular structures is ensured. At this point it is critical to obtain anterior-posterior (AP) and lateral images of the injured and adjacent segments to ensure appropriate radiographs can be obtained intraoperatively. If adequate images cannot be obtained, repositioning must be performed. We often use an OSI radiolucent flat-top surgical table because this allows the patient to be flipped to the Jackson table for the posterior approach without having to move the patient to a third bed. For the posterior portion of the procedure the Mayfield tongs can be attached to this bed.

Positioning on the Jackson table requires the pads be placed appropriately and checked by the surgeon. The head should be in a neutral position, with the eyes level and forward. After taping the shoulders in a depressed position radiographs should again be obtained before sterile preparation and draping. There are two key determinations that should be made radiographically prior to continuing the procedure. First, the cervical spine must be positioned anatomically as this is the position the fusion will assume. Second, it is prudent to make sure the anterior instrumentation and graft material remain in excellent position. Adjustments to the position can at this point be made through the Mayfield tong attachment.

STERILE PREPARATION AND DRAPING

Once the patient has been positioned and the C-arm is in place, sterile preparation and draping can proceed. It is critical that the surgeon be attentive to sterile technique and require it from the operative team. A wide prep should be made with the chief resident's maxim "never prep yourself out of a case" being good to remember (Fig. 23-2). We find most violations of sterile technique occur during the draping of the fluoroscopy unit. The surgeon must be facile with draping the C-arm and watch the operative team closely. For the posterior approach, it may be necessary to shave the patient's hairline to ensure adequate prep and secure attachment of drapes. One should ensure that all surgical instruments attached to the drape (cauterization units, suction devices, etc.) have enough throw to reach all portions of the surgical site and also ensure the functionality and positioning of pedals that will be needed during the procedure.

ANTERIOR APPROACH

We find a standard anterior approach to the cervical spine using a horizontal incision located in a skin fold is adequate for most single-level burst fractures (see Fig. 23-2). If a right-sided approach is used, attention must be paid to the location of the recurrent laryngeal nerve, which has a less consistent course on
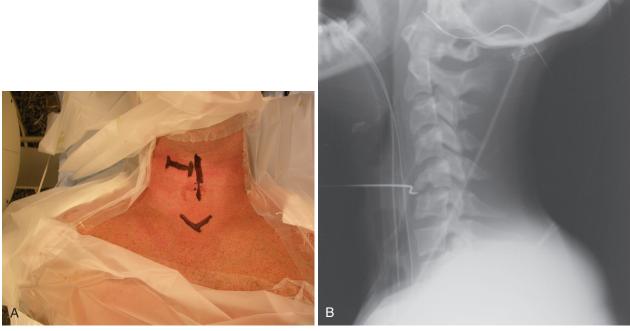
Fig. 23-2 *A,* Appropriate draping with skin marking for a right-sided C5 approach. Note position of incision in skin fold. *B,* Intraoperative localization lateral radiograph demonstrating bent spinal needle in disk space.

this side than on the left. When this structure is encountered it must be carefully retracted with minimal tension. If the patient has been previously operated in the neck region, direct laryngoscopy should be performed to confirm function of the recurrent laryngeal nerves. If a nonfunctioning nerve is detected, surgical dissection should be conducted ipsilateral to the nonfunctioning nerve.

Aggressive use of Bovie cauterization during the anterior approach may obscure tissue planes during the approach, and burning the dermis may lead to wound healing difficulties. We prefer to use the scalpel and blunt dissection. The appropriate surgical level is often easily identified because of hematoma and irregular bone morphology from fracture; however, it is prudent to confirm the location of surgery radiographically with a bent needle placed into the disk space (see Fig. 23-2). After self-retaining retractors have been placed, the balloon on the breathing tube can be deflated and then reinflated to decrease the incidence of tracheal mucosal injury, although there is conflicting evidence of the benefit of this maneuver. Before beginning the corpectomy, one should make sure the approach and retraction are truly complete. The esophagus is the most important at-risk structure during the approach and decompression of the anterior cervical spine. It is vital to always know its course and position in the surgical field. This ensures appropriate retraction and protects it from injury by surgical instruments, especially the high-speed drill. Next, the surgical microscope is positioned and adjusted for maximum visualization of the surgical site.

VERTEBRAL CORPECTOMY AND NEUROLOGIC DECOMPRESSION

An anterior corpectomy should be performed of on the fractured vertebral body. This begins with diskectomies at the level above and below the fracture. We use an 11-blade scalpel to perform a rectangular annulotomy and then pituitary rongeurs and Karlin micro-curettes for removal of the nucleus pulposus and endplate cartilage. After removal of the anterior annulus and the entirety of the nucleus pulposus the corpectomy is performed. This is accomplished using a combination of a high-speed drill and Lexel rongeurs. It is important to collect as much of the fractured vertebral body as possible during removal as it can be used for graft material. A frequent technical error during this portion of the procedure involves pushing on the fractured body and increasing retropulsion of bone fragments posteriorly into the SAC. This can potentially cause a SCI where none previously existed or exacerbate an incomplete SCI.

The posterior longitudinal ligament (PLL) narrows mid-body and widens at the disk space. The surgeon must be careful laterally mid-body as the dura mater and neurologic elements can become quickly exposed and potentially damaged here. Generally, bone should be removed until complete direct visualization of the dura mater is possible from the superior endplate of the inferior noninjured vertebral body to the inferior endplate of the superior noninjured vertebral body. Often the gentle convexity of the anterior spinal cord can be appreciated, affording the surgeon confirmation that

the corpectomy has proceeded far enough laterally. A blunt, angled nerve hook should be used to confirm that no bony or soft tissue fragments are causing foraminal impingement and nerve root compression. Decompression should similarly be confirmed posterior to the superior and inferior uninjured vertebral bodies.

The surgeon must be cautious not to proceed too far laterally at the superior and inferior corners as the vertebral arteries can be injured here. If preoperative imaging demonstrates significant comminution around the vertebral artery foramina, it can be useful to obtain a vascular imaging study to evaluate the patency of the vertebral arteries, and evaluate for rupture. Fracture or iatrogenic injury to a vertebral artery can be very stressful for the surgeon, and life threatening for the patient. The anesthesia team should be immediately notified of the potential for massive blood loss. Careful evaluation postoperatively should be made to diagnose potential cerebrovascular injury. Repair of the injured vessel rarely is possible. However, hemostasis can usually be obtained using a combination of thrombin surgical gel foam, direct compression with surgical paddies, bipolar cauterization, and use of coagulant application such at Tisseal (Baxter, Vienna, Austria). Time seems to pass very slowly after discovery of a significant arterial injury, but the surgeon must be patient with application of direct pressure for at least 5 minutes. Use of the operating room clock will help avoid premature reexposure of the injury.

It is not uncommon to encounter violation of the dura and arachnoid mater resulting from laceration by the fractured bone. A cerebrospinal fluid (CSF) leak may be encountered early in the corpectomy process if this is the case. Management of cervical dural tear should be performed with direct repair if possible. However, it may not be possible to identify the location of the dural leak, and some leaks may be irreparable. In this case a lumbar intrathecal CSF drain may need to be placed to decrease intradural pressure, and the leak is directly managed by use of coagulant application such at Tisseal (Baxter, Vienna, Austria).

PLACEMENT OF STRUCTURAL GRAFT

Once a thorough decompression has been undertaken and confirmed, the end-plates can be prepared and the structural graft can be placed. As previously stated; the vertebral endplates are sloped necessitating the removal of the anterior lip of the inferior endplate of the vertebra above the injured segment and the posterior lip of the superior endplate of the vertebra below the injured segment. Only a small amount of bone need be removed to square the endplate, usually on the order of 2 to 3 mm. The cartilaginous cap of the endplate is removed and the remainder of the endplate is prepared with the high-speed drill such that punctate bleeding is observed. Overly aggressive decortication of the endplates may result in unroofing of the underlying cancellous bone. This may lead to decreased structural properties and graft subsidence postoperatively. The appropriate-sized graft is selected by

choosing one with a snug fit that will correct cervical kyphosis. Traction can be applied through the Mayfield tongs to assist with the process. Next, the wound is copiously irrigated with 500 to 1000 ml of sterile irrigant. Success has been described with many different graft types and remains the surgeon's choice. We prefer to fill the selected graft and the lateral recesses around the edges of the graft with the local bone taken during the corpectomy, ensuring the graft does not impinge on the now decompressed neural elements.

PLATE FIXATION AND WOUND CLOSURE

With the structural graft in place, plate fixation is performed. The plate size selected should allow for screw fixation into the vertebral bodies above and below the injured level just deep to the endplate. Plates that are too long require dissection, which can injure the disk complexes above and below the injured levels leading to adjacent level ossification disease. The plate should be well-centered and the screws should be unicortical. We often use screws 14 mm in length, but screw size can be selected based on preoperative films. Usually the plate will have to be contoured to obtain a low-profile fit. We prefer to use plating systems that allow for some subsidence of the graft during healing so that compression by gravity can take place without failure of the plate system. Confirmation of the screw length and plate position as well as the location of the structural graft should be performed radiographically at this time. Finally a layered closure of the anterior wound is performed with a deep drain placed along the anterior plate. The patient is then repositioned, prepped, and draped for the posterior approach.

POSTERIOR APPROACH AND LATERAL MASS FIXATION

A standard midline approach to the surgical spine is performed. It is important to stay directly midline as this decreases intraoperative bleeding. Exposure should be performed only at the levels to be fused, and the surgeon should avoid injury to the facet joints superior and inferior to the levels required for fusion. Radiographic localization should be performed to confirm surgical level. It is important to evaluate preoperative radiographs to confirm the integrity of the bony posterior elements. Unrecognized lamina fractures can lead to inadvertent dissection into the canal and neurologic structures. If there is compression of neural elements or entrapment of the dura mater by posterior structures, a laminectomy can be performed (see Fig. 23-1). However, this is not usually the case and we prefer to leave the lamina intact as it provides a broad surface area for decortication and fusion. We prefer to use lateral mass screw and rod systems for fixation. We position and direct lateral mass screws according to the technique of Magerl.[22] This allows for longer screws, less variation in screw length, and generally a safer screw trajectory than other methods.[23] Screws are usually placed from the contralateral side of the operating table. Next, the wound is copiously irrigated with

500 to 1000 ml of sterile irrigant. After placement of surgical hardware we decorticate the lamina and lateral masses. Fluoroscopic images are taken at this time to confirm the position of the operative hardware.

The spinous processes of the injured, superior and inferior levels are resected and used for local autogenous graft material. The remaining bone from the anterior corpectomy can also be used at this time. We rarely find it necessary to harvest iliac crest bone graft for this portion of the procedure but it, or recombinant human bone morphogenetic proteins, can extend the local autogenous graft. Occasionally a small amount of graft extender such as allogeneic cancellous cubes or demineralized bone matrix products are used. Finally, a layered closure of the posterior wound is performed. Unless the subfascial portion of the wound is unusually bloody or a laminectomy has been performed we usually just place a single extra-fascial drain. The procedure is not complete until postoperative radiographs are carefully examined to confirm restoration of anatomic alignment of the cervical spine and appropriate position of operative instrumentation, as well as the structural graft.

CONCLUSION

Cervical burst fractures are complex injuries often complicated by SCI. They are frequently occurring injuries that can result from either low- or high-energy mechanisms. Addressing cervical spine instability is paramount when dealing with these injuries. Neurologic decompression may decrease the morbidity of SCI and allow some functional recovery of the injured elements. Most of the surgical techniques necessary for treatment of cervical burst fractures are familiar to the surgeon who treats other disorders of the cervical spine. A team approach is necessary for successful management of patients with SCI. The famous bank robber Willie Sutton was once asked why he robbed banks. He replied "that's where the money is." For the spine surgeon treating a cervical burst fracture, it is important to recognize where the money is. In terms of neurologic decompression and restoration of anatomic alignment of the spine, it is in the anterior approach. In terms of rigid stabilization, which allows for aggressive postoperative rehabilitation and predictability of fusion, it is in the posterior approach. Regardless of the outcome of SCI, patients with a cervical burst fracture have sustained a life-changing injury and require the guidance, honesty, and empathy of a confident and technically skilled surgeon to guide them through days, weeks, and months following their injury.

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C H A P T E R

24

HAREL DEUTSCH,

PRAVEEN MUMMANENI

Distraction-Extension Injuries of the Cervical Spine

INTRODUCTION

Neck hyperextension is a common mechanism of injury in cervical spine trauma in the adult and pediatric population.[1] Hyperextension injuries are common in motor vehicle accidents and falls. In motor vehicle accidents, the head strikes the dashboard or steering wheel and is driven backwards. Facial fractures are commonly associated with hyperextension injuries. In the pediatric population, sports injuries are also a common mechanism of injury. Hyperextension injuries are associated with "spear" tackling in football and rugby injuries.[2]

The most common types of cervical spine injuries noted with hyperextension injuries include traumatic spondylolisthesis of the axis (C2), extension vertebral body teardrop fractures, and posterior laminar fractures (Table 24-1). Neurologic involvement is rare in lamina fractures or in traumatic C2 spondylolisthesis. Disruption of the anterior column with rupture of the anterior longitudinal ligament (ALL) is seen with severe distraction-extension injuries. Neurologic injury is commonly associated with ALL rupture.

In the pediatric population, there are fewer fractures in children younger than 10 years compared with children 10 to 16 years old. Younger children have greater mobility of the spine and ligament laxity resulting in a lesser tendency to sustain cervical fractures.[3]

TRAUMATIC SPONDYLOLISTHESIS C2 (HANGMAN'S FRACTURE)

Traumatic bilateral spondylolisthesis of C2 is associated with hyperextension cervical spine injuries (Fig. 24-1). These fractures are also known as hangman's fractures. Hadley and Sonntag[4] reported 27% of all adult axis fractures are traumatic C2 spondylolisthesis. When accident reports were available, only 1 of 30 patients with C2 fractures involved in car accidents was wearing a seat belt. Unlike adults, pediatric C2 fractures are rare. Sun et al.[5] noted that in 71 consecutive pediatric patients with occipito-cervical spinal trauma, there was no incidence of either C1 or C2 fractures. Only several case reports of pediatric traumatic spondylolisthesis exist in the literature.[6,7] Halo vest immobilization is an effective treatment for pediatric C2 spondylolisthesis.

Adult traumatic C2 spondylolisthesis may be treated using rigid immobilization in a halo vest with excellent results.[8,9] A rigid cervical collar has also been shown to be effective in management of traumatic C2 spondylolisthesis.[10] Halo vest immobilization for C2 fractures in the pediatric population also has excellent results.[11] Surgical intervention is necessary in fractures with significant displacement or instability. Anterior C2-C3 fusions and placement of C2 pars screws have been described.[12]

POSTERIOR LAMINAR FRACTURES

Lamina fractures or lateral mass fractures are a common finding in hyperextension cervical injuries. Isolated fractures are treated with a rigid cervical collar. Neurologic involvement is rare.[13] Cervical lamina fractures may also be associated with more extensive anterior fractures or ligamentous destruction (Fig. 24-2). If a fracture extends through the transverse foramina, the possibility of a vertebral artery dissection must be kept in mind. Posterior lamina fractures are rare in the pediatric age group.

VERTEBRAL BODY FRACTURES AND ANTERIOR LONGITUDINAL LIGAMENT DISRUPTION

Hyperextension injuries are associated with fractures of the posterior third of the vertebral body. A posterior teardrop fracture may be seen and posterior displacement of a fragment may result in neurologic deterioration (Fig. 24-3). Rupture of the ALL is possible in severe hyperextension injuries. Disk space widening is noted on imaging (Fig. 24-4). Neurologic injury is commonly associated (Fig. 24-5). Vaccaro et al.[1] reported a

TABLE **24-1**   Common Spine Injuries in
                 Hyperextension Injuries

Traumatic spondylolisthesis of the axis (C2)
Vertebral body teardrop fractures
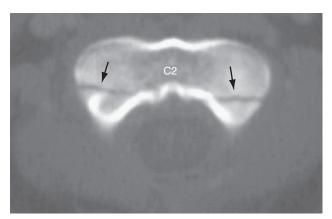Posterior laminar fractures

Fig. 24-1 Traumatic spondylolisthesis of C2. Arrows show the bilateral pars fractures on this axial CT scan.
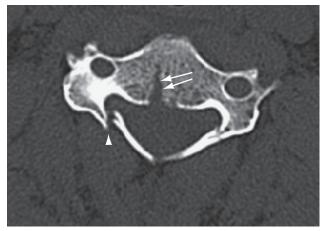

Fig. 24-2 Lamina fracture and posterior vertebrae fracture. The axial CT scan demonstrates a lamina fracture *(arrowhead)* and a posterior vertebrae fracture *(arrows)*.

Fig. 24-3 Posterior teardrop fracture. The sagittal CT reconstruction shows a C4 posterior teardrop fracture *(arrows)* with a retropulsed fragment.
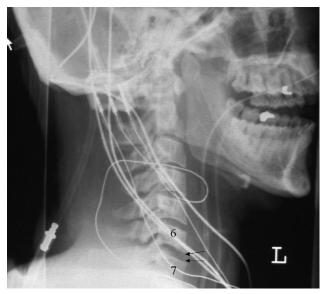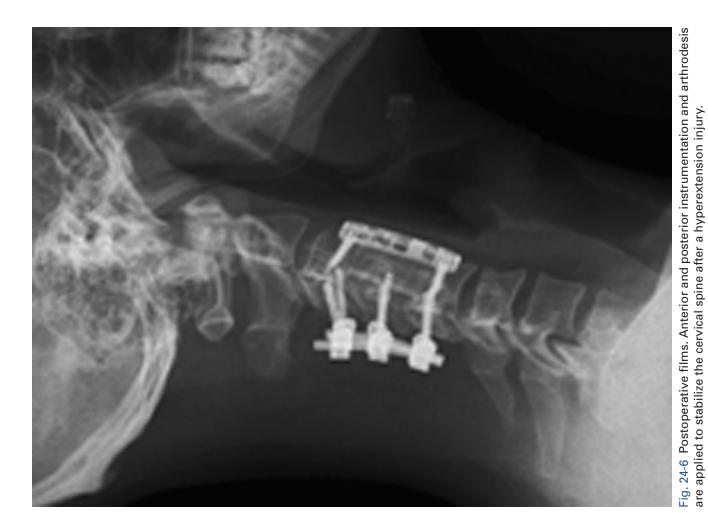

Fig. 24-4 Disk space widening. The lateral x-ray demonstrates widening of the disk space at C6-C7 with ALL rupture *(arrows)*.

high incidence of neurologic deterioration secondary to intra-operative overdistraction. Therefore, cervical traction and intra-operative manipulation should be applied carefully. Cervical stabilization is achieved preferably though an anterior cervical approach, instrumentation, and decompression. The instability noted in ALL rupture combined with posterior lamina fractures may require a combined anterior and posterior instrumentation (Fig. 24-6).

Fig. 24-6 Postoperative films. Anterior and posterior instrumentation and arthrodesis are applied to stabilize the cervical spine after a hyperextension injury.

Fig. 24-5 Spinal cord injury. The T2-weighted MRI shows a C4 vertebral body fracture and increased signal within the spinal cord opposite the C4/C5 interspace after a hyperextension injury.

CONCLUSION

Distraction-extension traumatic cervical injuries are common. Common mechanisms of injury include automobile accidents, falls, and sports injuries. The usual spectrum of injuries includes lamina and spinous process fractures treated with a rigid cervical collar. Neurologic injury is rare in isolated lamina or spinous process fractures. Traumatic C2 spondylolisthesis is well recognized with distraction-extension injuries and is usually effectively treated with a cervical collar or halo-vest immobilization. Finally, posterior teardrop fractures and rupture of the ALL are often associated with neurologic injury and cervical instability. Anterior column injuries with hyperextension injuries often require operative management. In the pediatric population, traumatic C2 spondylolisthesis is rare. Cervical fractures in general are seen less often in the pediatric age group, specifically in patients younger than 10 years.

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Lateral Mass Fractures of the Cervical Spine: Diagnosis and Surgical Management

INTRODUCTION

The diagnosis and management of cervical subaxial lateral mass fractures (C3 to T1) include a wide spectrum of injuries and associated clinical sequelae that are complicated by imprecise terminology and little agreement regarding optimal treatment techniques. Fractures of the subaxial lateral masses include minor undisplaced articular process injuries and complete fracture dislocations with gross instability and spinal cord injury. Treatment recommendations for these injuries range from external brace immobilization to combined anterior and posterior surgical stabilization.[1–3]

EPIDEMIOLOGY

Cervical spine injuries occur in 2% to 3% of patients with blunt trauma who undergo imaging studies[4,5] and in up to 6.7% of spine injuries.[6] Existing studies often have broad and nonspecific inclusion criteria, and most are limited to retrospective data from single institutions. Furthermore, lateral mass injuries often are associated with other, more significant, injuries and hence are not the primary focus of a significant number of articles.

Some incidence data can be found in the National Emergency X-Radiography Utilization Study (NEXUS),[4] a multicenter prospective observational study of the validity of a simple clinical-decision instrument for identifying patients who need radiographs after blunt cervical trauma. Of 34,069 patients with blunt cervical trauma, 818 (2.4%) had 1496 distinct cervical spine injuries.[7] Of the 785 fractures that occurred in the subaxial spine (C3 to C7), 30% involved the vertebral body, 15% involved the lateral mass/facet, 16.4% involved the lamina, and 5.9% involved the pedicle. The lateral mass/facet was most commonly fractured at C6 (38.5%) and C7 (25.6%).

Unilateral and bilateral injuries occur in approximately equal proportions, although some authors who carefully assess both lateral mass articulations have reported up to 75% bilateral injuries.[6,8] Injuries to the contralateral facet capsule and ligaments often are not recognized, and careful review of reformatted paramedian computed tomographic scans and magnetic resonance images is required. Subtle contralateral ligament injuries are more common when the ipsilateral facet is dislocated and subluxed to a high degree and when more sensitive imaging, such as magnetic resonance imaging (MRI), is used.[9,10] Injury of the contralateral facet complex has been observed in 63% of cases in which ipsilateral pure dislocation was noted, compared with 28% of cases with ipsilateral facet fracture.[11]

Lateral mass fractures often are associated with subluxation or dislocation and are considered to be within the same continuum of injury.[12,13] In association with rotational injuries of the facets, with which the subluxation often is more easily identified on radiographs than is a fracture, facet fractures have been noted in 54% to 78% of patients, with the remainder having pure ligamentous injuries.[8,11,13,14] When a fracture is present, the inferior facet is involved in 45% of cases, the superior facet of the subjacent vertebral body in 31%, and both facets in 24%.[11] The majority (79%) of fractures are vertically orientated through the lateral mass.[11] Isolation of the lateral mass (fracture of the ipsilateral pedicle and lamina) has been observed in 17% to 19% of patients.[11,13] This fracture separation of the lateral pillar is unique radiographically, clinically, and regarding treatment because this injury causes incongruence of the facet articulation at two adjacent spinal segments (Fig. 25-1). The implications of this form of fracture-separation of the lateral mass have been described by several authors.[11,13,15,16]

In addition to associated injuries at the same level, multilevel contiguous and noncontiguous injuries must be considered.
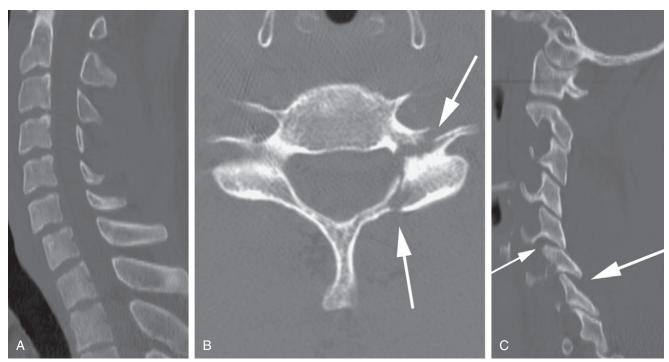
Fig. 25-1 *A,* Lateral view computed tomographic scan reformatted midline image shows subluxation at the C6-C7 interspace. *B,* Axial view image shows fractures through both the pedicle and lamina of C6 *(arrows),* leading to a fracture separation of the lateral mass or a "floating lateral mass." *C,* Paramedian reformatted image shows loss of congruence of both the C5-C6 and the C6-C7 facet joints *(arrows).*

Lateral mass fractures often are associated with noncontiguous spine injuries.[2,6,8,11,14,17–19] In a series of 3537 patients with blunt trauma who underwent spiral computed tomography (CT) at a single center, Brown et al.[20] noted a 19% incidence of patients with spinal injury at more than one spinal region. Bohlman,[21] in his review of cervical fractures, identified a 16% incidence of noncontiguous cervical spine injuries.

NEUROLOGIC INVOLVEMENT

Associated neurologic deficits can range from 5% to 37% in general series[12,22] and up to 96% in series in which nerve root deficits have been carefully recorded.[6,11,19] Cord injuries have been reported by a number of authors as occurring in 37% to 45% of unilateral facet injuries, with up to 15% of the injuries being classified as American Spinal Injury Association type A (complete).[1,12,23] A significant cord injury is more likely when the mechanism is pure flexion or when the facet is dislocated as a result of a pure ligamentous injury.[11,12,23] Most authors report satisfactory resolution of the nerve root deficit with timely reduction, decompression, and stabilization.[23,24]

Although injuries to the vertebral artery have been reported in association with cervical subluxations and dislocations, they rarely are associated with unilateral facet fracture/subluxations.[25,26] Propagation and displacement of a fracture into the transverse foramen have been reported as being associated with intimal vertebral artery injury. Although some studies have identified up to 45% of patients with cervical subluxations as having vertebral artery injuries, few if any of them present with symptoms.[25–28] Whether these patients require treatment for their concomitant arterial injury remains controversial.[27] We do not treat asymptomatic complete arterial occlusion.

CLASSIFICATION OF LATERAL MASS FRACTURES

No single cervical spine classification has been widely accepted, particularly regarding facet and lateral mass fractures. Classifications can be based on the morphology or the mechanism of the injury. The most commonly used classification is that described by Allen et al.[17] Although the Allen system classifies the majority of lateral mass fractures as distractive flexion or compressive extension, it does not fully account for those injuries that clearly occur as a result of coupled vectors in other planes, specifically in rotation. The Allen system classifies purely ligamentous unilateral facet dislocations as distractive flexion stage 2 (DF-2), whereas it classifies bilateral facet dislocations as DF-3 and facet fractures as compressive extensions. We have noted above that between one half and three quarters of rotational facet injuries have associated fractures, and one must question the relevance of a classification system that places these two injuries in exclusive and different classes.

Morphologic classifications tend to describe three categories of injury: pure ligamentous facet injury (subluxation or dislocation), combined fracture and subluxation or dislocation, and fracture occurring alone.[11,29] Others have added the unique pattern of fracture separation of the articular pillar (see Fig. 25-1).[13,15,16] Morphologic descriptions can further specify the percentage apposition of the superior and inferior facets, which of the facets is fractured, whether the injury is unilateral or bilateral, and the degree of subluxation as a percentage of the vertebral body diameter. The measurements have recently been standardized and described in detail.[30]

MECHANISM OF INJURY

The mechanism involved in injuries to the lateral pillar and facet is controversial. Fractures seem more likely to be caused by extension-compression, whereas ligamentous injuries seem to be the result of a rotation and flexion mechanisms.[17,18,22,31] Clinical and radiographic reviews have suggested that a combination of flexion and rotation is necessary to produce unilateral facet fracture/subluxations.[11–13,32,33] Some authors have described the mechanism of unilateral facet dislocation as occurring primarily with flexion in combination with axial rotation.[17,31,33] Some authors agree that rotation is a necessary element of injury mechanism,[34,35] whereas some have emphasized the importance of lateral bending in association with flexion and/or rotation.[33,34] Unilateral facet injuries have been created in human cadavers after compression and flexion loads and after axial rotation with or without lateral bending.[23,32,34]

Based on the literature and our experience, several features of the mechanism of injury seem to contribute to varying degrees. The magnitude, direction, and duration of any applied force are important. The order in which sequential forces are applied, the duration of the forces, and the length of any lever arm (eccentricity) have an impact on the final injury pattern. Finally, the coupled motions in the cervical spine can result in varying degrees of rotation and translation as a result of what seems to be a pure moment applied in a single direction.

In support of the various proposed mechanisms of injury are MRI findings that indicate injury to almost all of the ligamentous structures in the cervical spine to varying degrees. MRI findings associated with lateral mass fractures have been reported to show "significant" signal change occurring in the anterior longitudinal ligament and disk in 76%, posterior longitudinal ligament in 35%, and interspinous ligaments in 12% of cases.[18] Halliday et al.[36] studied unilateral facet fractures and noted injuries to the anterior longitudinal ligament (50%), posterior longitudinal ligament (29%), and interspinous ligaments (75%) shown by MRI. Regarding the unique fracture separation of the articular pillar, several injury mechanisms have been proposed, including hyperflexion-rotation in 81% of cases, hyperflexion-distraction in 14%, and hyperextension-rotation in 5%.[16]

CLINICAL PRESENTATION

All patients with suspected or confirmed cervical injuries need to provide detailed histories that include information on mechanism of injury and need to undergo physical examinations that include detailed neurologic examinations according to the International Standards for Neurological Classification of Spinal Cord Injury of the American Spinal Injury Association.[37] Cervical injuries often are diagnosed late because of the wide spectrum of clinical presentation, ranging from minor neck pain or no symptoms at all to major spinal cord injury. Various authors have reported that 33% to 40% of facet injuries are diagnosed late.[21,38,39] Associated injuries to the head and neck have been reported to contribute to the delay in diagnosis in up to 40% of facet injuries.[21] The remainder of the missed or delayed diagnoses are missed or delayed because of subtleties in the clinical and radiographic presentations or because of the presence of distracting injuries (usually orthopaedic), noncontiguous spine injuries (in 16%),[21] or inadequate radiographic evaluation.

Other than traumatic torticollis that can occur in patients with unilateral locked or dislocated lateral mass injuries, no other unique clinical features of lateral mass fractures compared with other subaxial cervical injuries are noted. The dominant presenting symptoms are neck pain and radicular pain with or without sensory/motor disturbance in the distribution of a single nerve root.

For patients with neurologic injury, carefully documented detailed neurologic examination is crucial to both acute and subsequent management decisions and strongly predicts the neurologic prognosis of the patient.[32] Numerous studies have shown that the degree and type of neurologic injury are important predictors of neurologic and functional recovery.[40,41]

DIAGNOSTIC WORKUP

In general, radiographic workup proceeds in a stepwise fashion, classically starting with appropriate plain radiography and then CT and/or MRI. The data obtained from these studies combined with the clinical presentation of the patient aid in decision making regarding overall management (operative vs. nonoperative) and the specific surgical treatment, if surgery is deemed necessary.

Two large prospective studies have generated simple decision instruments designed to reduce patient risk and the cost of unnecessary investigations.[4,42] With the NEXUS low-risk criteria[23] tool, patients must meet five criteria to be classified as having a low probability of injury and thus avoid the need for cervical radiographs. The Canadian C-spine rule[42] is based on three high-risk criteria, five low-risk criteria, and the ability of patients to rotate the neck. Both decision instruments have emphasized the importance of identifying risk factors related to mechanism of injury in an attempt to improve the pretest likelihood of identifying

unstable injuries (and indirectly avoiding investigations into minimal injuries).

Stiell et al.[43] showed that for alert patients with blunt cervical trauma who are in stable condition, the Canadian C-spine rule is superior to the NEXUS low-risk criteria regarding sensitivity and specificity for cervical spine injury. This structured and evidence-based approach to obtaining cervical spine radiographs will likely lead to a higher proportion of cervical facet injuries undergoing radiographic evaluation at the initial medical encounter.

Once investigation is deemed necessary, plain radiography historically has been the mainstay of initial radiographic assessment for facets suffering from trauma (Fig. 25-2, *A*). Plain radiography, however, often is inadequate and can miss 50% to 60% of injuries.[44,45] For this reason, and with the advent of more efficient helical CT, many high-volume trauma centers have used helical CT as the primary mode of assessment for spinal column injuries (Fig. 25-2, *B*).[20,45] This method has been shown to be clinically more accurate and cost-effective.[46] In a recent study, McCulloch et al.[44] found that plain radiography missed 48% of injuries and helical CT missed only one minor injury (2%).

Looking specifically at lateral mass injuries, multiplanar (axial, sagittal, and coronal) CT provides a reliable means for the assessment of lateral mass fractures, particularly for nonradiologists.[47] Furthermore, helical CT is performed in rapid scan time, provides high-quality multiplanar reformatted images, and also has the added benefit of imaging the occipitocervical and cervicothoracic junctions, thus also reducing the incidence of missed injuries in those two regions. We think that helical CT from the occiput to T3 should be the radiographic investigation of choice in the evaluation of blunt cervical spine trauma that warrants radiographic investigation based on the Canadian C-spine rule.

For patients with neurologic injury, MRI is the test of choice for assessing neural compression and spinal cord injury (i.e., directly visualizing and differentiating spinal cord hemorrhage and edema) (Fig. 25-2, *C*).[9,10] Furthermore, MRI plays an invaluable role in the assessment of soft tissue injury (disk and ligaments) and can be very influential in the assessment of stability.[18,36] Typically, the use of fat suppression techniques enables visualization of the associated soft tissue injuries with more reliability. For neurologically intact patients with kyphosis or splaying of the facet joints shown by CT, assessment of the soft tissue posterior ligamentous complex is recommended.

CLINICAL MANAGEMENT

NONOPERATIVE TREATMENT

Isolated lateral mass and facet fractures, minor fractures, and minimally displaced fractures can be treated nonoperatively with a short period of immobilization (up to 6 weeks) in a cervical orthosis. Depending on the energy of the injury and its degree of displacement, collar treatment with close observation typically is adequate for low-energy undisplaced facet or lateral mass fractures. Upright plain radiographs should be obtained with the patient in the brace before discharge. The patient should be seen 1 to 2 weeks after injury, and repeat upright radiographs should be obtained to rule out any evidence of subluxation.

Repeat clinical and radiographic assessment is conducted at 2, 4, and 6 weeks after injury. If after 6 weeks of immobilization in an orthosis, the upright radiographs show no change in alignment or displacement, controlled flexion-extension views can be obtained. Characteristically, the fracture remains visible radiographically (plain radiographs and computed tomographic scans) long after solid union is achieved. Occasionally, bone is seen bridging the interbody space anteriorly. If the patient's clinical symptoms change or if instability is suspected based on the radiographs at any time, repeating CT and considering MRI (if neurology or occult ligamentous injury is an issue) is indicated.

Multilevel fractures with little or no evidence of soft issue injury are more likely to be treated nonsurgically because of the extensive fusion necessary to achieve surgical stability. Halo vest immobilization has been used in managing multilevel fractures, although the reported failure rate is significant.[12,31,48–50] It is hoped that nonoperative treatment of these multilevel injuries will allow maintenance of motion and negate a potentially morbid multilevel fusion.[48]

Halo vest treatment, historically the mainstay of cervical fracture treatment, is not without complications.[12,49,50,51] A higher risk of failure with nonoperative management is noted for patients with more than 40% vertebral body collapse, more than 10 to 15 degrees of kyphosis, subluxation over 15% of the body diameter, and incongruent facet reduction.[6,7,14,38,39,51,52] One of the aspects of performing evidence-based medical practice is to consider patient preference. In our practice, halo thoracic vest immobilization, particularly when compared with the outcomes of single-level anterior or posterior cervical fusion, is not easily accepted by patients.

A number of authors have proposed an association between malalignment of the fractured vertebrae and poor clinical outcome.[38,39,51] Rorabeck et al.[39] identified 20% of their nonoperatively-treated patients who developed spontaneous fusion and reported that those whose fractures remained displaced had a higher degree of pain. Similar results of a Mayo clinic study were reported; 42% of the nonoperatively treated patients had pain and stiffness, whereas only 10% of the operatively-treated patients reported similar symptoms.[38] Preliminary results of a multicenter study (performed by the Spine Trauma Study Group) of facet fractures treated both surgically and conservatively suggest that although the initial results of both treatment methods are similar, over the longer term, the conditions of the nonoperatively-treated patients deteriorate regarding pain and function.[53] This might be because of the onset of secondary degenerative changes in the nonsurgically-treated patients.
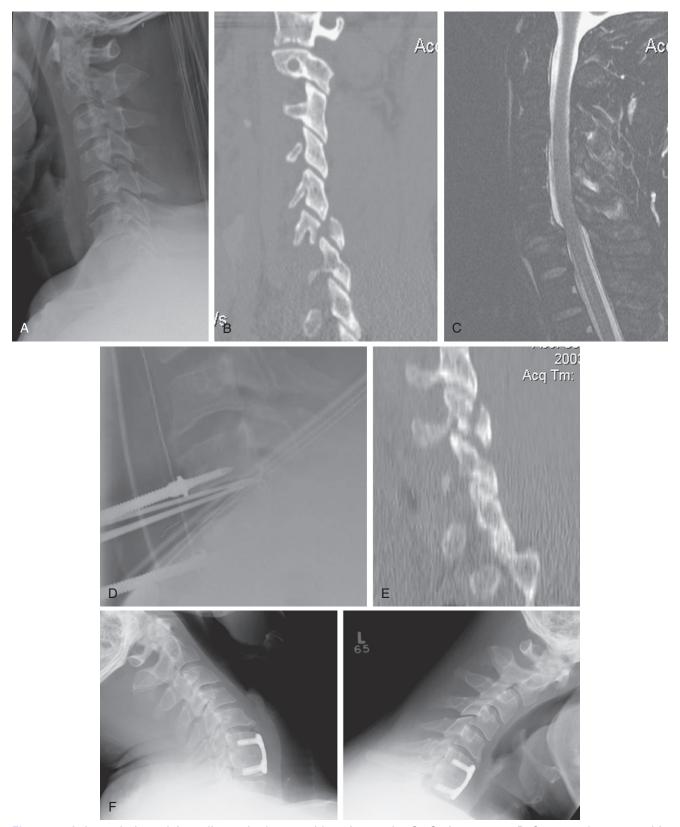
Fig. 25-2 *A,* Lateral view plain radiograph shows subluxation at the C6-C7 interspace. *B,* Computed tomographic paramedian reformatted image reveals a vertical lateral mass fracture. *C,* Fat-saturated magnetic resonance image confirms the degree, location, and extent of disk disruption. *D,* During intraoperative reduction via an anterior approach, particularly at the cervicothoracic junction, the use of small nerve hooks can assist in visualization and confirm adequate reduction. *E,* Postoperative computed tomographic scan confirms reduction and alignment. *F,* Three-month postoperative flexion and extension views confirm solid bony union.

The application of tongs for traction, reduction, and stabilization of lateral mass fractures is not routinely required.

OPERATIVE TREATMENT
INDICATIONS

Although historically, controversy has existed regarding the need to reduce dislocated cervical facets, all acute dislocated injuries should be reduced and stabilized. Any dislocation that presents beyond 2 weeks after injury will likely fail nonoperative closed reduction and is best treated with open reduction and fusion, whereas injuries that present beyond 4 to 6 weeks will require a formal circumferential release and/or osteotomy. On occasion, a facet fracture dislocation that presents beyond 4 to 6 weeks might be left as it is, depending on the degree of symptomatology, stability of the injury, and patient preference.

For patients with radiculopathy, several authors identified dramatic improvements in neurologic symptoms and signs (root pain and function) as soon as compression of the nerve root was relieved.[24,54] If complete or incomplete spinal cord injury is present, the degree of instability is likely best treated surgically.[2,54]

The displaced traumatic separation of the articular pillar is a particularly unstable injury that requires surgical treatment in all cases.[15,16,55] This injury affects two adjacent motion segments (Fig. 25-1, *C*), although the intervertebral subluxation usually affects only the lower level.[16] Although most authors recommend two-level (disk) posterior symmetrical fusion, we have had success with single-level anterior diskectomy, fusion, and plate fixation at the subluxed (usually the lower) level, as long as an anatomic reduction is achieved.

CONTRAINDICATIONS

Surgical stabilization is contraindicated for those patients who are unable to tolerate anesthesia. In a case in which the area of incision has been compromised by burns, tracheostomy, previous radiation, and so forth, surgery might not be advisable.

OPERATIVE PROCEDURE AND TECHNIQUE

OPERATING ROOM SETUP

An anesthesiology staff, including respiratory therapists who are trained and educated in dealing with unstable cervical spine injuries, is required. From an equipment perspective, a spinal trauma table that enables patients to be rotated from a supine to a prone position, when required, while maintaining traction on the cervical spine is essential (e.g., Jackson Spinal Table, OSI Products, Union City, CA; Stryker operative wedge frame, Stryker, Kalamazoo, MI). Gardner-Wells tong traction should be applied in the operating room with the patient under local anesthesia to provide intraoperative spine stabilization, if the tongs have not already been applied for attempted closed reduction or stabilization before surgery. Furthermore, a radiolucent table facilitates the use of C-arm fluoroscopy or other intraoperative imaging modalities. Oblique fluoroscopy views might provide the best imaging of the cervicothoracic junction. From an anesthetic perspective, patients with facet injuries typically require awake fiberoptic intubation while the cervical spine is controlled and stabilized.

OPERATIVE PROCEDURE

An anterior, posterior, or combined approach can be used to address lateral mass fractures of the cervical spine (Table 25-1).

POSTERIOR APPROACH

For the posterior approach, patients are positioned prone and intraoperative images are obtained to confirm that no change in the alignment has occurred. The arms typically are tucked at the sides and the shoulders taped down as necessary. To avoid upper brachial plexus palsy, care should be taken not to aggressively pull down on the shoulders. Allowing the shoulders to fall forward somewhat might aid intraoperative radiographic or fluoroscopic visualization. A standard midline posterior exposure is used.

Great care must be taken not to expose the facet capsule or interspinous ligaments of the adjacent levels above and below the intended levels of fixation. Often, only two thirds of the lateral mass must be exposed to adequately obtain localization for safe lateral mass fixation (center of the lateral mass). The level above or below can be used to estimate the center of the lateral mass, where only partial exposure is performed.

If the injured spinal levels are translated, reduction is required. Such injuries can be associated with a posterior hematoma, which, if present, should be evacuated before reduction. For superior facet fractures (that have not dissociated from the inferior facet or rotated within the foramen), reduction of the translation by use of the spinous process above and below (ensure that no fracture of the associated lamina is present) is performed. Symmetrical alignment of the dorsal elements is directly visualized and can be confirmed with imaging. An oblique ("pillar") view can improve visualization of the lateral mass complex. For inferior facet fractures with impaction or dislocation of the remaining inferior facet ventral to the superior facet, reduction is greatly facilitated by partial removal of the cephalad portion of the superior facet as required. A gentle levering of the remaining unfractured inferior facet over the superior facet with the use of a Penfield 4 dissector also can facilitate reduction. When using the latter techniques, however, distraction should be minimized; partial bony removal of the facet

TABLE 25-1   Surgical Approaches to Subaxial Cervical Facet and Lateral Mass Fractures/Subluxations

APPROACH	INDICATIONS	ADVANTAGES	DISADVANTAGES
Anterior	SCI with anterior spinal cord compression, particularly when disk protrusion is identified on MRI Unilateral facet injury ± radiculopathy Bilateral facet injury (minor fracture in one or both facets) ± radiculopathy	Quick, simple, and well-tolerated approach Eliminates the specialized equipment, time, and risk associated with the prone position in the unstable C-spine Low infection rate and improved correction of sagittal alignment	Risk (albeit very low) of airway, esophageal, or vascular injury Open reduction can be challenging for less experienced surgeons High failure rate when performed alone in the presence of facet or endplate fractures
Posterior	Unilateral or bilateral facet injury: Decompression if required is achievable by reduction High risk of failure with ACDF alone (endplate/body fracture, comminuted facet fractures) Rigid spine (e.g., ankylosing spondylitis) requiring multisegmental fusion Cervicothoracic junction	Directly addresses facet pathoanatomy Biomechanically superior fixation compared with anterior alone Technically familiar approach for surgeons Easily extensile to adjacent segments	Increased time and risk associated with prone positioning Increased disruption of paraspinal muscles and adjacent levels Higher infection rate than anterior surgery Fracturing of facets may limit segmental fixation and may necessitate fusion of additional motion segments
Combined	Any of the posterior indications where an anterior decompression is also required To supplement either anterior or posterior fixation when concerns remain regarding stability, and it is desirable to avoid multilevel fixation	Circumferential stabilization and decompression (if required) Improved stability in high-risk cases Advantage of anterior decompression combined with improved posterior stabilization	Combined approach may require postoperative ventilation because of neck swelling (airway control) Longer surgical time with need for repositioning Anteriorly-placed bone graft may displace during transfer to prone position for posterior fixation

MRI, magnetic resonance imaging; SCI, spinal cord injury.

will help to achieve this. Physiologic lordosis can be achieved by gentle extension of the cervical spine or can be facilitated through instrumentation with slight compression across the injured segment, either directly on the screws or via a wire or cable between the adjacent spinous processes or obliquely from a spinous process to an inferior facet. Decortication of the facet joints and insertion of local bone should be performed before this.

Most centers currently use a cervical rod screw fixation system, with lateral mass fixation at C3 to C6 and pedicle screw fixation at C7 and T1 (Fig. 25-3) (please see the chapter on posterior cervical instrumentation). When the lateral mass is comminuted to a degree whereby posterior lateral mass fixation is not possible, the fixation might have to extend across the injured level (i.e., a two-motion segment fusion is required). To address any concerns regarding stability, either fixation can extend to adjacent levels or concomitant anterior fusion and plating can be performed.

Traumatic separation of the lateral mass requires rigid fixation spanning the "floating" lateral mass. Injuries at the

C7 or T1 facets with intact pedicles can be stabilized by using direct pedicular fixation at the injured level. For injuries with associated anterior column bony deficiency or for which lordotic alignment cannot be achieved, we recommend fixation two levels above and below the injury. For patients with osteoporosis and for patients with rigid spinal segments above and/or below the injuries, such as ankylosing spondylitis or diffuse idiopathic skeletal hyperostosis, multilevel fixation (two to three levels above and below) should be applied (see Chapter 63).

ANTERIOR APPROACH

The anterior approach typically is used if anterior cord compression caused by disk or bony pathologic abnormality is present and requires removal before a reduction maneuver can be performed. Some surgeons prefer the anterior fixation technique for all cases.[3,8,56] The anterior approach, when appropriate, eliminates the potential risk of injury during turning to the prone position (Table 25-1). The patient is left supine with the head held in an adjustable head holder (Mayfield horseshoe head holder with tong traction).
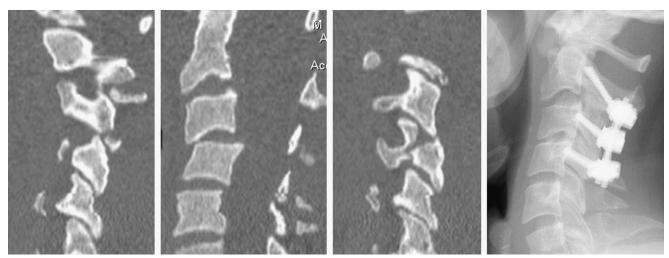
Fig. 25-3 Computed tomographic reformatted sagittal view images of C2, C3, and C4 lateral mass fractures with subluxation and kyphosis at the C3-C4 interspace. Posterior open reduction and segmental instrumentation facilitates the restoration of stability, lordosis, and alignment.

A standard anterolateral Smith-Robinson approach is used. If significant disk injury is present and/or translation exists, the injured level is readily identifiable. Intraoperative images are obtained to confirm the level. The disk is removed. Before removal of the disk, unicortical distraction pins can be placed into the vertebra. The placement of the pins parallel to the endplate and in the midsagittal plane of the individual vertebra might facilitate reduction (Fig. 25-2, D). After the posterior longitudinal ligament has been resected and the dura has been decompressed, reduction should be performed. With comminuted facet fractures, an increased incidence of loss of reduction is associated with anterior plating alone[8] and care must be taken that the segment does not sublux or malrotate (see Fig. 25-2, D). Adequate lordosis must be confirmed and usually is achieved by extending the patient's neck (after decompression and reduction are achieved) by lowering the head holder toward the floor. A trapezoidal anterior interbody graft and an anterior plate are then placed. One should note that the anterior approach should not be used as a stand-alone approach in the presence of endplate fracture or for very comminuted facet fractures (Fig. 25- 4).[8] Postoperative CT can be used to confirm reduction (Fig. 25-2, E). If anatomic alignment is not achieved or comminuted posterior element injury with gross preoperative translation is present, combined anterior and posterior fixation should be considered. After 4 to 6 weeks of activity restriction, flexion and extension lateral views should confirm radiographic union (Fig. 25-2, F).

COMPLICATION AVOIDANCE

Avoidance of neurologic and vascular complications associated with instrumentation placement requires thorough knowledge of the relevant anatomy and instrumentation techniques (see Chapters 14 and 15). Placement of pedicle

screws at C7 or T1 can be facilitated by a laminoforaminotomy and the use of intraoperative fluoroscopy or surgical navigation. Fixation failure can be minimized by taking into account the presence of osteoporosis and stiff or rigid motion segments above or below the level of injury and by ensuring appropriate lordotic alignment and augmentation by using interspinous wiring or by adding a level of fixation above and/or below for the aforementioned scenarios. Appropriate selection of anterior, posterior, or combined surgical approaches is based on the fracture pattern, neurologic involvement, and surgeon's experience and preference and can avoid fixation complications in many instances (see Fig. 25-4). Furthermore, adjacent segment instability caused by iatrogenic facet destabilization should be avoided.

SURGICAL OUTCOMES

Patients with cervical facet fractures, regardless of the method of treatment, do not necessarily return to generic or disease-specific functional status of the normal population. Although fusion rates with anterior or posterior surgery are high (>90%) and objective results of neurologic examinations return to normal, often some degree of residual pain and stiffness is present and affects the patient's generic health-related quality of life.[3] Reported 36-Item Short Form Health Questionnaire and North American Spine Society/American Academy of Orthopaedic Surgeons cervical follow-up outcome scores do not necessarily return to those of a healthy normative sample.

CONCLUSION

The diagnosis and management of cervical subaxial lateral mass fractures (C3 to T1) address a wide spectrum of injuries that account for approximately 15% of cervical spine injuries.

Fig. 25-4 Computed tomographic scan *(A)* and magnetic resonance image *(B)* reveal a complex bilateral facet fracture dislocation with a concomitant endplate compression fracture of C7 *(white arrow* in *B)*. Because of spinal cord injury and extruded disk material, anterior surgery was performed. Postoperative images *(C)* reveals that the subluxation was not fully reduced *(arrow)*, and based on both the facet fractures and the endplate fracture, construct failure *(D)* could have been predicted. Salvage required combined anterior (corpectomy) and posterior surgery *(E* and *F)*.

The injuries typically occur from flexion or extension mechanisms with varying degrees of rotation and/or shear. Associated clinical symptoms vary from minor neck pain to complete quadriplegia. Obtaining a detailed history and performing a physical examination are essential to assessment. Multiplanar computed tomographic assessment is the diagnostic test of choice; however, MRI might assist in the evaluation of neurologic symptoms and the presence or degree of ligamentous or disk injury when suspected. In general, isolated lateral mass/facet fractures, minor fractures, or minimally displaced fractures can be treated nonoperatively with a short period (≤6 weeks) of immobilization in a cervical orthosis. The decision to operate and the operative approach and technique cannot be standardized to a single approach and require that patient factors, injury mechanism, pathoanatomy, and surgeon factors be carefully considered. For displaced injuries or those with neurologic (root or cord) compromise, operative intervention tends to result in better outcomes. Overall, the outcomes for subaxial lateral mass fractures are good; however, regardless of the method of treatment, these injuries often are associated with some degree of residual pain and stiffness, which affects patients' overall quality of life.

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Surgical Complications Related to the Management of Traumatic Spinal Injuries of the Cervical Spine

INTRODUCTION

Traumatic spinal injuries occur most commonly in the cervical region of the spine. Before the development of modern spinal instrumentation, surgical decompression and attempted reconstruction was fraught with complications secondary to ongoing spinal instability. Although instrumentation has helped reduce serious complications such as graft displacement, resubluxation, and pseudarthrosis, the surgical treatment of these traumatic injuries may have unique and increased complications in comparison to other elective cervical spine procedures.

In this chapter we review the surgical complications related to the treatment of traumatic cervical spine injuries. Although there is a large body of data addressing surgical complications in cervical surgery, firm conclusions are sometimes difficult to ascertain from the literature, especially regarding traumatic injuries because there are few studies evaluating surgical complications in the setting of cervical spine trauma. Most studies evaluating surgical complications include several indications for surgery, of which trauma patients are a single subset. We consider *general complications* that can occur with any cervical spine procedure for trauma and also *surgery-specific complications* associated with a particular surgical procedure.

GENERAL COMPLICATIONS

Some complications are universally seen in all types of cervical spine procedures independent of the specifics of the surgical approach. In the setting of surgery for cervical spine traumatic injuries, general complications would include postoperative wound infections, potential for neurologic decline, and morbidity related to the harvesting of autograft bone.

POSTOPERATIVE WOUND INFECTIONS

Postoperative wound infection after spinal surgery is a relatively uncommon entity occurring in approximately 1% to 5.4% of all spinal surgeries[1–7] and in 0% to 7% in cases of anterior cervical plating.[8–14] This unfortunate complication has significant consequences, such as prolonged hospitalization, increased costs, and poor patient outcomes. Although the overall rate of infection after spinal surgery is relatively low, certain procedures and patients are associated with a much higher risk of infection. It has been well documented that posterior approaches to the spine have exponentially higher infection rates than anterior approaches,[3] and the use of spinal instrumentation has also been associated with increased risk of postoperative infection.[6,15] Traumatic spinal injuries have been noted to have higher rates of infection than do equivalent elective procedures.[16,17] Rechtine et al.[17] noted a 10% postoperative infection rate in 235 patients with traumatic injuries of the thoracic and lumbar spine and also noted statistically significantly higher risk of infection in patients with complete neurologic injuries. There are a multitude of potential causes for increased infection rates in trauma patients. Tissue disruption and edema increase the probability of seroma and hematoma collections, which can subsequently become infected. In addition, trauma patients commonly have skin abrasions, other surgical wounds, prolonged periods of bed rest, external bracing over the incisions, prolonged stay in the intensive care unit, propensity for other infections, and long complicated surgical procedures, which all likely contribute to higher infection rates. Clinical suspicion for infection should be elevated in the postoperative trauma patient that

exhibits prolonged wound drainage, fevers, and increasing pain around the surgical site. Evaluation is typically performed with a thorough examination assessing wound appearance, vitals signs, laboratory studies (white blood count, C-reactive protein, and Westergren sedimentation rate), and contrast-enhanced magnetic resonance imaging (MRI) if possible or computed tomography (CT). There should be a low threshold for surgical exploration with wound culturing. Treatment typically consists of surgical washouts, that is, debridements, and antibiotic therapy.

INTRAOPERATIVE NEUROLOGIC DECLINE

Surgical procedures on patients with cervical spinal cord injury (SCI), especially incomplete injuries, have potential to cause intraoperative neurologic decline. An injured spinal cord with areas of tenuous perfusion may be prone to further damage with fluctuations in blood pressure with anesthetic exposure or any minor manipulation with surgical decompression. Given these risks, the proper timing of surgery after incomplete cervical SCI, especially central cord injuries, is a topic of substantial debate.[18] Some authors argue that surgery should be delayed in the setting of an incomplete SCI, when instability and canal compression are not a concern, to avoid medical complications. Mirza et al.[19] retrospectively compared early (less than 72 hours) and delayed (greater than 14 days) surgery in 43 patients with acute SCI and found no significant difference between groups when looking for pulmonary, neurologic, thrombotic, wound-related, or infectious complications. Vaccaro et al.[20] performed a prospective comparison of early (less than 72 hours) versus late (greater than 5 days) surgery for cervical SCIs and found no difference between groups in terms of acute postoperative intensive care unit stay, neurologic improvement, or inpatient rehabilitation duration. Finally, an evidence-based review of decompression surgery for acute SCI found no convincing evidence that surgical complications are increased with early surgery.[21] The timing of surgery after cervical SCI does not appear at this time to affect the complication rate if there is appropriate vigilance by anesthesia and surgical staff. Intraoperative spinal cord monitoring is recommended, both motor-evoked potentials and somatosensory-evoked potentials, to help reduce the risks of decompression and stabilization. Mean arterial pressure should be regulated at greater then 85 mmHg throughout the preoperative, intraoperative, and initial postoperative periods in all patients with SCI. The anesthesia staff should be reminded of optimal blood pressure guidelines preoperatively and to check the blood pressure before any corrective maneuvers or decompression of stenotic levels.

ILIAC CREST BONE GRAFT (ICBG) HARVEST

Harvesting of donor bone from the iliac crest for arthrodesis procedures is very common owing to ease of access and availability of large quantities of both cortical and cancel-

lous bone. Although use of iliac crest autograft is considered the gold standard for spinal fusions, it is associated with significant complications at a rate of 10% to 49%.[22–26] Pain is the most common complication of ICBG harvest, with chronic pain reported in 10% to 39% of cases.[26–34] Other complications include pelvic fractures (Fig. 26-1), meralgia paresthetica, superior cluneal and ilioinguinal nerve injuries, hematoma, and infection.[26,28,29,33,35–40] A retrospective questionnaire-based study of 134 patients who underwent ICBG harvesting revealed impairments in ambulation (12.7%), recreational activities (11.9%), work activities (9.7%), activities of daily living (8.2%), sexual activity (7.5%), and household chores (6.7%).[33] With these significant complications, many centers have reduced the use of autograft bone in favor of allograft as much as possible, except in situations in which pseudarthrosis is a significant risk, such as in smokers and revision surgeries.

SURGERY-SPECIFIC COMPLICATIONS

ODONTOID SCREW FIXATION

Anterior odontoid screw fixation is a common technique for treating Anderson type II and shallow-type III odontoid fractures.[41–43] This technique is favored by some surgeons for odontoid fracture stabilization because it has the potential to preserve motion at the atlantoaxial articulation, in contrast to treatment via posterior atlantoaxial arthrodesis. The most common complications with this approach are hardware failure and dysphagia. Hardware failure may be resulting from screw breakage or pullout and has been reported to occur in 2.6% to 10% of cases.[41,42,44] Subach et al.[45] reported that elderly patients and patients with osteoporosis experience

Fig. 26-1 Right pelvic fracture *(arrow)* resulting from the harvesting of posterior iliac crest bone graft.

more screw pullout because of their poor bone quality and resulting weak screw purchase. Apfelbaum et al.[41] reported hardware failure, with either odontoid screw breakout or pullout, in 10% of 117 patients. They reported that many of the breakouts were at the anterior border of C2 at the head of the screw and retrospectively noted that most of the breakouts were associated with type III fractures in which, in hindsight, there was too significant damage of the body of C2 to secure a screw. In instances of screw backout, Apfelbaum et al. reported that most of the cases of backout were associated with inadequate capturing of the distal odontoid cortex with screw threads (Fig. 26-2). Strategies to prevent hardware failures include (1) placing the starting hole 2 to 3 mm posterior to the anterior body of C2 to provide a good margin of cortical bone to prevent anterior breakout and (2) engaging the screw

in the distal cortex of the odontoid, by drilling and tapping through the distal cortex, to prevent screw backout. Anterior oblique fractures, in which the fracture slopes down from a dorsal to a ventral direction or posterior superior to anterior inferior (Fig. 26-3), are treacherous fractures to treat using any modality. In our experience, these fractures have a tendency for the odontoid process to slide forward or sublux on the body of C2, even after stabilization procedures. Apfelbaum et al.[41] reported higher rates of failure with odontoid screw fixation in these oblique fractures.

Dysphagia is a common complaint after odontoid screw fixation and appears to be especially prevalent in elderly patients. Henry et al.[44] reported a very low incidence (2.5%) of dysphagia in a retrospective review of 81 patients (mean age = 57 years) treated with odontoid screw fixation. However, Dailey et al.[46] reported results more comparable to our experience, with dysphagia rates of 30% in patients older than 70 years and noted the symptoms typically improved after temporary treatment with a feeding tube. Other potential
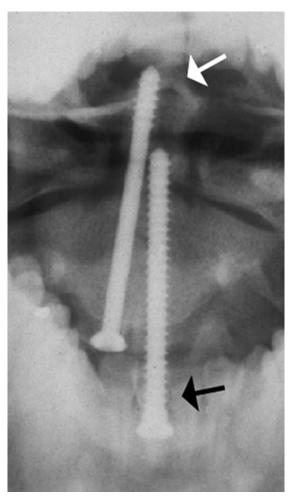

Fig. 26-2 Example of an odontoid screw which has backed out because of poor distal screw purchase from failure to engage the screw in the cortex of the odontoid tip. The screw on the left is in appropriate position with threads in the distal odontoid cortex *(white arrow).* The screw on the right (black arrow) has backed out approximately 1 cm because of failure to engage screw threads in the distal cortex.
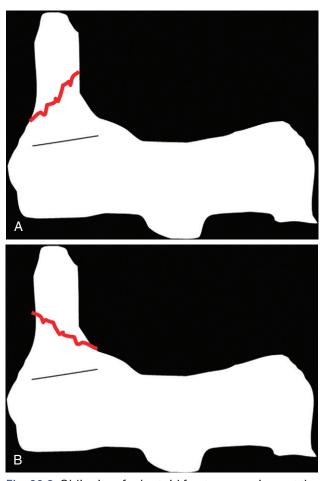

Fig. 26-3 Obliquity of odontoid fractures can impact the success of treatment with anterior oblique fractures being especially difficult to treat with external stabilization and odontoid screw fixation. Anterior *(A)* and posterior *(B)* oblique fractures are demonstrated in this illustration.

complications include recurrent laryngeal nerve (RLN) and superior laryngeal nerve (SLN) injuries, which may be more common in odontoid screw placement than in other subaxial approaches given the high dissection. Although they have not been published, we have heard of devastating complications with K-wire–based odontoid screw placement, including impaction of the K-wire into the brainstem, and for this reason many surgeons prefer noncannulated systems.

POSTERIOR ATLANTOAXIAL ARTHRODESIS PROCEDURES

Odontoid fractures, unstable Jefferson fractures, and other complex fractures of C1 and C2 may be treated with posterior atlantoaxial arthrodesis. Early procedures for atlantoaxial arthrodesis, such as the Gallie, Brooks, and Sonntag techniques, used wire fixation of an autograft bone between the posterior arch of C1 and spinous process of C2.[47–49] Subsequent advancements with screw fixation, initially atlantoaxial transarticular screw fixation and then C1-C2 polyaxial screw-rod fixation, provide more immediate stability than wiring techniques alone, which has reduced the need for postoperative halo immobilization and improved fusion rates to greater than 90% in most studies.[42,50–54] Primary complications with these procedures have included wound infections, vertebral artery injuries, hardware failure, autograft morbidity, and pseudarthrosis.[50–52,55]

Vertebral artery injuries are potentially the most devastating injuries associated with screw fixation techniques. Strokes may result from unilateral arterial injuries, and death has been reported from bilateral vertebral artery injuries during posterior atlantoaxial arthrodesis procedures.[52,56] The vertebral artery is most vulnerable to injury at the pars articularis of C2, where it can occasionally loop medially into the pars (Fig. 26-4). Transarticular screw fixation, in which a screw is placed through the pars interarticularis of C2, across the C1-C2 lateral mass joint, and into the lateral mass of C1, has a higher risk for vertebral artery injury than C1-C2 polyaxial screw-rod techniques that place a shorter screw in C2. Madawi et al.[57] reported that a "high-riding" vertebral artery preventing safe transarticular screw placement is present in nearly 20% of patients. Gluf et al.[52] reported vertebral artery injury in 2.8% of transarticular screw cases from a series of 198 patients. Wright and Lauryssen[56] reported vertebral artery injury in 3.9% of 1058 patients having transarticular screw placement from pooled data of 92 surgeons, but only two patients had neurologic deficits from vertebral artery injury. Although associated with a lower risk for vertebral artery injury, the use of a long or low C2 pars screw or low-medial C2 pedicle screw can result in arterial injury. In situations in which vertebral artery injury is suspected because of the presence of pulsatile bleeding, it is recommended by many surgeons to place the screw to tamponade the bleeding. If vertebral artery injury is suspected during the placement of the first of two planned transarticular screws, the contralateral transarticular screw should not be
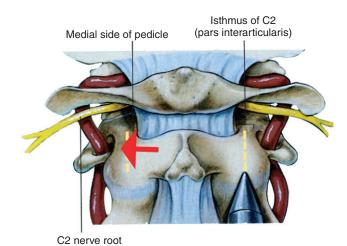

Fig. 26-4 The vertebral artery is vulnerable to injury at the pars interarticularis of C2 as demonstrated in this drawing *(red arrow).* The vertebral artery may loop medially into the pars and be injured during screw placement.

placed. A unilateral screw system will suffice in these situations. The use of postoperative CT angiography is recommended when arterial injury is suspected. Complete vertebral artery occlusions are usually tolerated by the patient, but dissections and other injuries may require antiplatelet therapies to prevent embolic strokes. Consultation with a neurovascular specialist to assist with management of vertebral artery injuries should be strongly considered.

Another complication that is unique to posterior atlantoaxial screw fixation is occipital neuralgia from C2 nerve root irritation. This condition is more commonly associated with C1-C2 polyaxial screw-rod fixation, but cases have been reported with transarticular screw fixation.[55] Stulik et al.[58] reported a 14% incidence of postoperative pain or paresthesias in a C2 distribution after C1-C2 polyaxial screw-rod fixation. With C1-C2 polyaxial screw-rod fixation, the C1 lateral mass screws are placed just above the exiting C2 nerve root. Irritation may be secondary to surgical manipulation of the nerve root or more commonly from chronic irritation from the adjacent screw. In attempt to reduce the incidence of this root irritation, smooth-shank screws, where only the distal 15 to 20 mm of the screw is threaded and the remainder of the shaft is smooth, have been recommended for this application. Some authors have recommended sectioning of the C2 nerve at the ganglion to facilitate placement of C1 screws and gain access to the C1-C2 lateral mass articulation for arthrodesis, although this technique is not yet widely accepted.[53]

ANTERIOR SUBAXIAL CERVICAL SPINE APPROACHES

The anterolateral approach to the cervical spine, medial to the sternocleidomastoid and carotid sheath, and lateral to the trachea and esophagus, was initially described for disk

removal and interbody fusion independently by Robinson and Smith and by Cloward in the 1950s.[59] Anterior cervical diskectomy and fusion, with or without corpectomy, has been successfully applied to trauma cases, and evolution of internal fixation plates has reduced complication rates. An anterior approach in trauma cases is advantageous for anterior neural decompression and reconstruction of the anterior spinal column.

Dysphonia is one of the most common complaints after anterior cervical surgery and in most instances is secondary to laryngeal edema from intubation and retraction that resolves after a few days. Other potential, and possibly more distressing, causes for dysphonia include injury to the *RLN* or SLN. Both of these nerves may be injured during the approach or as a result of stretch or compression with retraction.[60–64]

The RLN supplies all of the laryngeal muscles, except the cricothyroid, and provides sensation to the larynx below the level of the vocal folds. Unilateral RLN injury results in vocal cord paralysis with symptoms of hoarse voice, vocal breathiness or fatigue, and possibly aspiration because of the inability to approximate the vocal folds.[62,65] RLN palsy after anterior cervical surgery occurs temporarily in 3% to 24% of patients.[65–67] In most situations (about 90% of cases), it is a neuropraxia that resolves within 6 to 12 weeks.[65,66,68,69]

The SLN supplies the cricothyroid muscle and provides sensation to the larynx structures above the level of the vocal folds. SLN injury does not cause vocal cord paralysis but can cause permanent voice changes and may have significant aspiration risks. Patients with a SLN injury typically have a "nasal" voice change and often have difficulty with oral secretions. Silent aspiration, because of loss of sensation above the vocal folds, is a major concern with SLN injury.

RLN injury after anterior cervical surgery has received more attention in the literature than SLN injuries. Common principles may be used to prevent both of these nerve injuries. Careful blunt dissection is recommended and avoidance of cutting deep structures that appear to be neural in origin is important. Revision surgeries have been shown to have higher risk of RLN and SLN injury because of the complexity of dissection through scar tissue.[65,70] The SLN is most vulnerable in upper cervical approaches, and the RLN can be visualized during low cervical approaches. The RLN has a more predictable course within the tracheoesophageal groove on the left side of the body. On the right side, the RLN takes a more oblique course in its initial ascent and it often lies outside of the tracheoesophageal groove during a large portion of its ascent. The combination of a more oblique ascent, less redundancy (shorter nerve), and a path outside of the tracheoesophageal groove leaves the right RLN more exposed, and thus, it has been theorized it may be more vulnerable to retraction and compression injury. For this reason, some anatomists and surgeons have speculated that a left-sided approach reduces the risk of RLN injury, but there are conflicting data regarding whether this strategy truly reduces RLN injuries. The authors of some studies have noted a higher incidence of RLN injury with a right-sided approach,[71,72] whereas the results of other series have shown no correlation between the side of approach and incidence of RLN injury.[70,73] In addition, a right-sided approach is more ergonomic for right-handed surgeons, which may provide for an overall safer surgery on the patient's right side.

Several mechanisms of RLN injury have been proposed, but results from experimental cadaver dissection by Apfelbaum et al.[65] suggest that most injuries result from compression of the nerve within the endolarynx when the retractor displaces the larynx against the endotracheal tube. In that study, the occurrence of RLN palsy decreased when cuff pressure was released and reset after retractor placement to allow the endotracheal tube to reposition itself within the larynx. Anchoring the retractors beneath the longus colli muscles may help minimize pressure on laryngeal structures and reduce RLN injury. Occasional release of the retractors will also reduce prolonged periods of pressure on the RLN. Intraoperative electromyographic (EMG) monitoring of the posterior pharynx has been used to monitor RLN activity, but no specific indicators of RLN injury were identified that could be used intraoperatively to make changes.[65,74]

For patients with prolonged symptoms of hoarse voice or aspiration, laryngoscopic examination can confirm a diagnosis of RLN or SLN injury. A unilateral RLN palsy will show the vocal cord in a fixed paramedian position on laryngoscopy, whereas a combined RLN and SLN injury will have the affected vocal cord in a paramedian but more lateral position. The cricothyroid muscle has an adducting effect on the cords and, when this is absent, as in SLN palsy, the cords assume a more lateral position. Evaluation of sensation above the vocal folds may help determine whether SLN injury is present. Postoperative laryngeal electromyography, with placement of an electrode in the thyroarytenoid muscle, has also been reported to help predict the likelihood for recovery with an accuracy up to 70%. Absence of voluntary motor activity, sharp waves, and fibrillation potentials on EMG are associated with a poor prognosis.[75]

When a RLN or SLN injury is confirmed, speech therapy, aspiration precautions or feeding tube, and otolaryngology evaluation may prove beneficial. Vocal cord injections with Gelfoam, Teflon paste, or adipose tissue have been used to help enlarge the paralyzed vocal cord to bring it to a median position, which allows the contralateral functioning cord to contact it. Vocal cord injections are very effective at preventing aspiration and restoring the voice quality to close to normal.[76,77]

Dysphagia is another common complaint after anterior cervical spine surgeries and in most situations is secondary to local tissue edema and is self limited. Other potential causes include graft protrusion, hardware protrusion, hematoma, infection, glossopharyngeal nerve injury, hypoglossal nerve injury, adhesion formation, and pharyngeal plexus denervation.[61,78–80] Dysphagia often occurs with dysphonia, suggesting

a common cause in many circumstances.[81] Reported rates of dysphagia after anterior cervical surgery vary from 1.7% to 60%.[73,79,80,82,83] A study of 71 patients who underwent anterior cervical diskectomy and fusion with a minimum follow-up period of 5 years found that dysphagia often does not resolve, with a rate of 35.1%.

Esophageal perforation is a rare, but very serious, complication of anterior approaches that most commonly presents with infection in the subacute postoperative period. Most injuries occur at the time of surgery, but delayed injuries as a result of hardware migration have been reported years after surgery.[84,85] Esophageal injuries have also been reported to occur at the time of initial trauma as a result of distraction injuries.[86] In a large retrospective series of 44 patients with esophageal injury after anterior cervical surgery, 77% had cervical spine fractures as the indication for surgery and 63% had instrumentation placed.[87,88] Common presenting symptoms included neck and throat pain, dysphagia, hoarseness, and aspiration initially with later findings of infection such as fever, wound changes, and sepsis. Patients may become critically ill with tracheoesophageal fistula and mediastinitis. With the rarity of infection in anterior cervical spine surgery, esophageal injury should always be ruled out as a potential source for infection. Esophagoscopy, Gastrografin swallow studies, and injection of indigo carmine dye into the esophagus at the time of anterior cervical wound exploration can all be used to find esophageal injuries.[87–89]

In a series of 44 patients with esophageal injury after anterior spine surgery, Gaudinez et al.[87] reported that hospital stays were uniformly long (mean stay = 253 days), and mortality was reported in 4.5% of patients. Treatment of esophageal perforations typically includes direct surgical repair of the perforation, drainage, and antibiotics. Surgery may consist of direct repair or more complex procedures involving placement of a vascularized muscle flap over the esophageal injury. Often times anterior instrumentation is removed in situations in which esophageal injury was confirmed.[87,88]

The use of anterior cervical plating in the setting of trauma has been reported in multiple case series.[8–14] Pseudarthrosis occurs in less than 5% of cases in most published case series,[8–10,13,14] but, in a retrospective study, Henriques et al.[12] reported nonunion in 25% of 36 patients with distractive-flexion injuries treated with anterior plate alone. A subgroup analysis found that the rate of pseudarthrosis correlated with the severity of injury. Stage 1 distractive-flexion injuries (flexion strain) had a 100% rate of solid union, which decreased to 88% with stage 2 injuries (unilateral facet joint dislocation) and 46% with stage 3 injuries (bilateral facet joint dislocations with severe posterior soft tissue injury). Furthermore, 5 of 7 patients with stage 3 injuries that failed anterior fusion alone had resubluxation within 48 hours from the time of surgery (Fig. 26-5). In a series of 92 patients, Ripa et al.[14] reported hardware malpositioning in 13% of cases, but screw loosening or hardware breakage generally is an uncommon event with reported rates between 0% and

7.1%.[8–11,14] Other reported complications associated with anterior cervical plating in trauma patients include dysphagia, transient Horner syndrome, pneumonia (6.7%), and deep venous thrombosis (1.7%).[9,14,90]

Corpectomy is often used in situations of trauma and has been noted to have higher complication rates than anterior cervical diskectomy and fusion surgeries. Overall complication rates in two large series evaluating anterior corpectomy with screw-plate fixation ranged between 21%[91] and 23%,[92] although only 25% and 21% of the patients treated had experienced trauma, respectively. General surgical complications were primarily comprised of dysphagia (7%–8%), infection (3%–4%), and cerebrospinal fluid leak (0%–7%). Hardware and graft complications included pseudarthrosis (1.2%–7.1%), telescoping of the graft (3%–4%), screw pullout (2%–8%), and plate migration (1%–2%). The most common medical complications were deep venous thrombosis (2%–6%), pneumonia (3%–7%), and death (2%–3%). In a large retrospective review of cervical trauma patients, including 56 who underwent anterior corpectomy with fibular strut graft, dislodgement of the graft occurred in 10% of cases at follow-up and some loss of initial reduction was found in 54% of cases.[93] Vascular injuries, with injuries to the vertebral arteries from wide corpectomy, have also been described to occur in up 2.2% of cases.[92,94] A case of internal carotid artery thrombosis after multilevel cervical corpectomy, which was attributed to stasis secondary to retraction, has also been described.[95]

Pediatric cervical spine injuries are rare in comparison to adult injuries, with a paucity of literature specific to this population. The surgical management of pediatric cervical spine injuries is limited to small case series[96–99] and case reports.[100,101] Brockmeyer et al.[96] performed 24 pediatric cervical spine internal fixation procedures, which included 20 traumatic injuries, with 12 anterior plates and 2 posterior plates. Two complications—one infection and one graft and hardware failure—occurred.

POSTERIOR SUBAXIAL CERVICAL SPINE APPROACH

Posterior approaches are often used for decompression, open reduction maneuvers, and stabilization of cervical spine injuries. Facet dislocations, which cannot be reduced preoperatively, are typically more easily reduced with a posterior approach than with an anterior approach. Advancements in spinal instrumentation with polyaxial screws and rods replacing plating and wiring techniques have improved fixation and reduced complications.[102]

Concerns about complications from posterior cervical constructs with screws and rods have focused on improper insertion of the screws. A review of 78 patients treated with lateral mass plates, including 11 with fracture/dislocations, found that 9% of patients had complications directly related to lateral mass screws insertion.[103] Screw complications included nerve root injury, facet violation, broken screws,
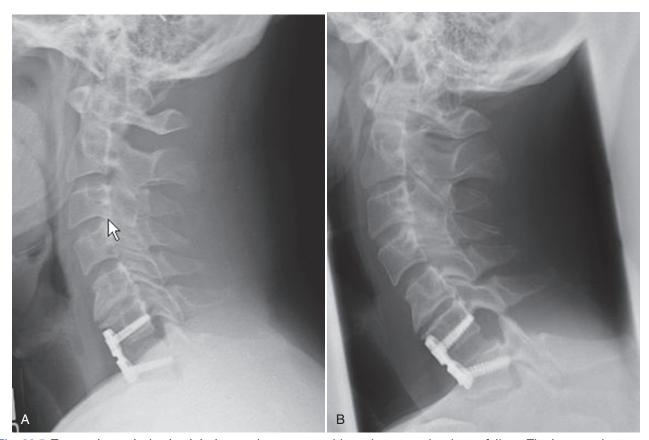
Fig. 26-5 Traumatic cervical spine injuries can be very unstable and prone to hardware failure. The images shown are from a case of bilateral C6-C7 jumped facets that was treated with reduction and *(A)* C6-C7 anterior cervical diskectomy and fusion (ACDF). *B,* The patient developed increasing neck pain 1 week after surgery and was noted to have resubluxation with perched facets at the instrumented level as shown. The patient was treated with posterior instrumented arthrodesis at C6-C7 without any further complications.

screw avulsion, and screw loosening, although each of these complications occurred with 1% or less of screws. Other complications, all arising in less than 3% of patients, included infection, pseudarthrosis, SCI, iatrogenic foraminal stenosis, lost reduction, and broken plates. In another series of 44 patients with posterior cervical plates, of which 42 were trauma patients, several complications were documented, including pseudarthrosis (7.3%), increased kyphosis (5.3%), infection (4.5%), and loose screws (3.8%).[102] Surgical revision was required in 7% of patients. Finally, the authors of a review of 180 patients who were treated with posterior cervical pedicle screw fixation, including 70 trauma patients, documented that screws violated the pedicles in 6.7% of patients resulting in one case of vertebral artery injury without neurologic injury and three patients with nerve root injury (1.7%).[104] In this series, infection (1.1%) and pseudarthrosis (0.7%) rates were both low.

Brodke et al.[105] performed a small prospective randomized trial comparing anterior and posterior approaches in cases of unstable SCI from C3 to C7. Overall, there were no differences in neurologic, kyphotic, or translation improvement. No significant differences in pseudarthrosis (10% and 0%), pain (35% and 26%), respiratory issues (10% and 4%), or instrumentation failure (5% and 4%) were observed in the anterior and posterior groups, respectively. Capen et al.[92] retrospectively reviewed 212 patients who underwent surgical stabilization after cervical trauma in an attempt to compare anterior and posterior approaches. Most complications, including deep venous thrombosis, pulmonary difficulties, and wound issues, occurred with similar frequencies between the two groups; however, the anterior approach was responsible for a higher rate of dysphagia and dysphonia (9%), as would be expected.

CONCLUSION

General complications common to all cervical spine surgeries for traumatic injuries include infection, pseudarthrosis, and hardware failure. Infections are notably increased for cervical spine surgery in trauma patients compared with nontraumatic indications and with posterior instrumentation. Harvesting of iliac crest bone for allograft is associated

with several potential morbidities including pain, pelvic fracture, hematoma, meralgia paresthetica, and nerve injury. Specific surgical approaches also have unique complications related to the involved anatomy. Posterior atlantoaxial arthrodesis is prone to vertebral artery injury in the vicinity of the C2 pars interarticularis, which can result in stroke with a unilateral injury or death with a bilateral injury. The anterior subaxial cervical spine approach is associated with injury to the SLN or RLN, potentially leading to dysphagia, changes in voice, or an increased aspiration risk. An uncommon but very serious complication of the anterior approach is esophageal perforation, which leads to prolonged hospital stays and potentially death. The potential complications of the aforementioned surgical approaches need to be kept in mind, but they should not deter the surgeon from appropriately using these procedures in the treatment of traumatic cervical spine injuries.

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Mechanisms of Injury in the Thoracic/ Lumbar Spine: Concepts, Pathomechanics, Classification, Instability, and Clinical Applications

INTRODUCTION

The diagnosis of thoracic and lumbar spinal injuries has been radically improved, and the treatment fundamentally altered, by the development and routine application of computed tomography (CT) and magnetic resonance imaging (MRI).

The development of surgical procedures, training, and implants capable of providing either anterior or posterior (or both) "short-segment instrumentation" for any spinal injury is a relatively recent development. These technologic advances have dramatically improved the functional outcomes for the unfortunate patients who require reconstruction of severe injuries.

Until the development of hospitals, trained surgeons, and spinal techniques that could provide "short-segment solutions," many fractures were managed nonoperatively. Prior to these developments, "long-segment solutions" involving considerable morbidity were the only surgical alternative and therefore were often avoided. At the present, at least in the first world, short-segment surgical solutions are commonly selected to manage spinal injuries that were formerly, and in the second and third world, still are treated nonoperatively.

Prior to the routine use of CT and MRI scanning, plain radiographs were the only available modalities to assess the amount of damage done to the spine during an accident. During this period, a voluminous literature developed in an attempt to describe which spinal fractures were "stable" and which were "unstable." This debate existed since surgeons were apparently unsure about the integrity of the spinal ligaments, amount of comminution, and inherent stability of the injured vertebrae.

This stable versus unstable characterization implied that "unstable injures" needed an operation to do well, whereas "stable injuries" could uniformly be treated nonsurgically and also do well. This stable versus unstable dichotomy was expected to provide the treating surgeon with an easy paradigm to guide treatment decisions.

Unfortunately, this simplistic analysis completely ignored the fact that spinal cord and spinal column injuries occur with an incredible array of clinical and structural variety. This diversity makes patient management a very delicate and challenging test.

The stable versus unstable debate has been "put to rest." The variety and complexity of spinal injuries has been clearly identified by the routine use of CT and MRI scanning in patients who are suspected of having a serious spinal column injury.

Routine whole-body CT scans are now performed in most American trauma centers for major trauma victims. Review of these scans quickly and accurately identifies spinal osseoligamentous injuries and provides critical information necessary for prompt and proper patient management. This imaging also provides a clear illustration of injuries that were formerly missed on plain radiographs, often providing a clear origin of lingering complaints, which were formerly dismissed.

MRI scanning of patients who are identified with spinal fractures accurately identifies existing ligament and soft tissue injuries and permits selection of appropriate operative or nonsurgical options, often within hours of the patient's accident. The development and widespread use of routine CT and MRI scanning, along with new surgical approaches and implant systems, has created a new era in the literature regarding spinal injury management, rendering all past literature clinically irrelevant.

CLASSIFICATIONS OF SPINAL FRACTURES

Many spinal injury classifications have been developed by authors prior to, and without the benefit of, CT and MRI scan information. These have been extensively catalogued and discussed in the literature.[1] After CT scanning was developed, the three-column classification of Denis[2,3] and McAfee et al.[4] was described. With this classification—and its arbitrary delineation of the spine into columns—came an undue emphasis on the "middle column." Middle column involvement was recognized and emphasized, to the point of ignoring the major contributions to spinal stability from the facet joints, disk, ligaments, and even the integrity of the remaining vertebral body.

The rest of the spine became ideologically less important in the determination of stability and surgical indications. Some surgeons even incorrectly proposed that any middle column involvement—no matter how trivial—was an indication for surgical fixation. Although classifications based solely on plain radiographs and CT images may be interesting on a historical basis, most have no value in current clinical use. Nearly all of these classifications attempt to characterize fractures as stable or unstable based solely on plain radiographic data. The array of spinal injuries is far too complex to permit such simplistic analysis.

At the present, two classifications: the *AO/ASIF* classification[5] (Fig. 27-1) and the load-sharing classification[6] (Fig. 27-2), use plain radiographic data combined with information from the CT scan to accurately characterize spinal fractures. These two classifications are complementary. The AO classification characterizes spinal injuries by the major injury vectors, which are suggested to have caused the injury-flexion, extension, or multiple vectors with translation. The load-sharing classification characterizes injuries according to the amount of comminution that has occurred at the injured vertebrae, regardless of the injury mechanism. This classification predicts how well the spinal column itself will transfer the load of the trunk, once a short-segment surgical repair is provided.

A newer classification proposed by Vaccaro et al.[7] classifies thoracolumbar injuries based on three major characteristics: bony morphology, the integrity of the posterior ligaments, and the neurologic status. A composite score from these three variables attempts to determine operative indications. MRI findings are the means by which the posterior ligaments are evaluated and scored. This is the first such classification to adequately incorporate the degree of soft tissue injury, as assessed by MRI, into the classification scheme. Although not an absolute guide of surgical indications, the thought processes this classification induces can certainly aid clinical decision making. These three classifications, used together, along with the patient's particular clinical and radiographic assessment, should provide all the information a clinician needs to treat their patients.

TRANSLATIONAL DISPLACEMENT

The existence of translational displacement in a spinal injury, even though minor, is extremely important. Translation implies a much more serious injury than injuries without translation (Fig. 27-3). Translation suggests the presence of bony comminution in all three columns of the spine, as well as at least minor stretching or rupture of most, if not all, spinal ligaments. This soft tissue injury is analogous to an appendicular dislocation. With a complete elbow or knee dislocation, often all the stabilizing ligaments are torn; and with translation, such is the case in the spine. The existence of translational displacement—even though minor—defines a "fracture-dislocation" in the load-sharing classification.[8] This is the most severe form of spinal injury—equivalent to a "type C" injury in the AO classification. Although spinal injuries with translation can be treated nonsurgically, the overwhelming majority of injuries with translation warrant surgery. However, this axiom only applies in patients who can tolerate surgical stabilization and in areas with medical facilities capable of providing such services.
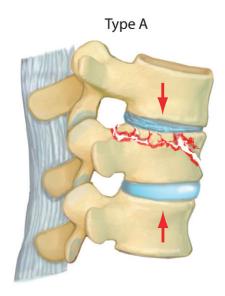
CLINICAL ASSESSMENT OF TRAUMA PATIENTS

Prioritizing the management of the spinal fracture in a multiply traumatized patient requires experience and judgment. The trauma center physician and surgeon staff must judiciously consult appropriate services, prioritizing the patient's injuries to preserve the patient's life and restore and maximize his or her function. This prioritization can only be done "on site" by physicians who have all pertinent information available to them.

Although there may be many competing priorities, all are important, but all must be "put in line" for treatment in a clinically appropriate order.

CLINICAL ASSESSMENT OF THE SPINE IN A PATIENT WITH A SPINAL FRACTURE

A fundamental "data point" in the evaluation of the multiply injured patient is the physical examination of the spine and the patient's neurologic examination.

When the patient is stable enough for a complete spinal examination, he or she should be log rolled to examine the entire spinal column. If a cervical collar was applied at the accident site, it should be cautiously removed and the spine examined carefully and gently for deformity, tenderness, and swelling. If the patient is symptomatic, CT scanning should be completed before range of motion testing is performed. The same basic approach should be followed in examining
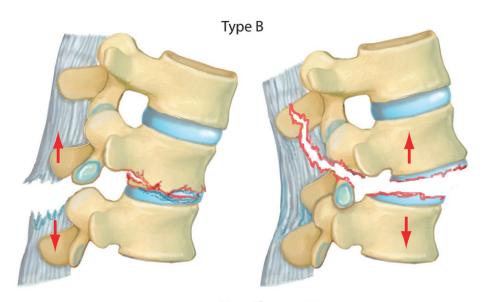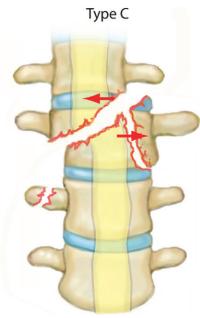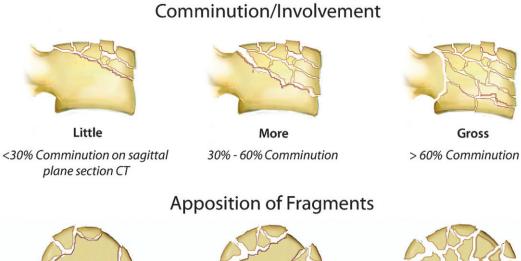
Type A

Type B

Fig. 27-1 AO/ASIF principles in spine surgery.

Type C

Comminution/Involvement

Little

<30% Comminution on sagittal plane section CT

More

30% - 60% Comminution

Gross

> 60% Comminution

Apposition of Fragments

Minimal
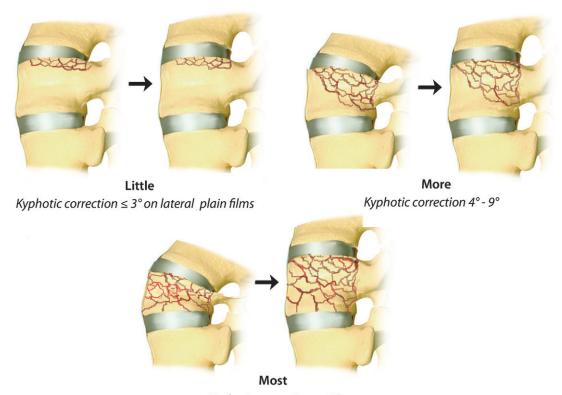
Minimal displacement on axial CT cut

Spread

At least 2 mm displacement of <50% cross section of body

Wide

At least 2 mm displacement of > 50% cross section of body

Deformity Correction

Little

Kyphotic correction ≤ 3° on lateral plain films

More

Kyphotic correction 4° - 9°

Most

Kyphotic correction ≥ 10°

Fig. 27-2 The load-sharing classification of spinal fractures. (From Parker JW, Lane JR, Karaikovic EE, Gaines RW: Successful short-segment instrumentation and fusion for thoracolumbar spine fractures. Spine 25:1157–1169, 2000.)

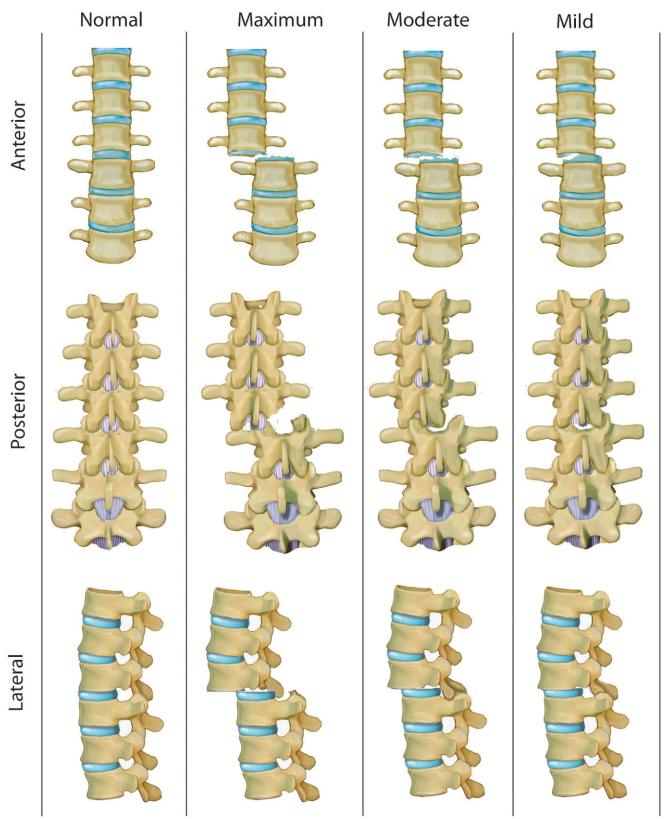Normal Maximum Moderate Mild

Anterior

Posterior

Lateral

Fig. 27-3 Anteroposterior, posteroanterior, and lateral views of translational displacement in a thoracolumbar fracture. The drawing on the left represents an intact spine, followed by drawings of translation from gross to subtle. (From Parker JW, Lane JR, Karaikovic EE, Gaines RW: Successful short-segment instrumentation and fusion for thoracolumbar spine fractures. Spine 25:1157–1169, 2000.)

the thoracolumbar spine. If the patient is on a backboard, he or she can certainly be log rolled and gently examined for deformity before CT scans are done.

A meticulous neurologic examination should also be performed, as thoroughly as the patient's clinical condition permits. Rectal examination for tone must be included, as well as detailed sensory testing, looking for the possibility of a "sensory level" and/or sacral sparing.

TREATMENT SELECTION FOR SPINAL INJURIES

Once the treatment of the spinal fracture reaches the highest "clinical priority," treatment selection begins. However, only after both clinical and radiographic examinations are completed and evaluated can treatment recommendations be made. No area of orthopaedics requires more judgment than selecting treatment for spinal fractures. Every spinal fracture can be treated operatively or nonoperatively. The selection depends on the availability of health care resources that are focused on this unique problem, as well as the overall well-being of the patient.

There are numerous physicians, surgeons, and medical centers where nonsurgical treatment has provided acceptable management of these injuries for many years. Centers experienced in nonoperative management, which do not have experienced surgical teams, should not be expected to provide the same quality surgical care as a center that has experienced surgical teams. Both operative and nonoperative centers focus their efforts on spinal alignment and healing, along with measures that facilitate recovery of a spinal cord injury, if such has occurred.

ROLE OF SURGERY IN IMPROVING SPINAL CORD FUNCTION FOLLOWING A SPINAL CORD INJURY

A voluminous literature on this topic has demonstrated the limited role of surgical management—acute or chronic—in optimizing recovery for patients who sustain a spinal cord injury.

Current recommendations for emergency surgery in the acute setting include only those patients who show progressive neurologic deterioration while they are under observation—an exceedingly limited number. The functional benefits of elective spinal cord decompression and spinal stabilization for patients with "incomplete" neurologic injuries have been well documented. However, operative intervention has not achieved a place in producing neurologic recovery for patients who have a "complete" lesion at the time of their injury. Nonetheless, patients with a complete spinal cord injury can benefit from surgical spinal column stabilization to hasten their rehabilitation and avoid post-traumatic spinal deformity.

ROLE OF SURGERY AND SPINAL STABILIZATION IN IMPROVING SPINAL COLUMN FUNCTION—AVOIDING "POST-TRAUMATIC KYPHOSIS" AND DELAYED SPINAL CORD COMPRESSION

There is general agreement that performing spinal stabilization procedures leads to better long-term spinal alignment in patients who have had severe spinal column injuries with or without spinal cord injury. Oner and colleagues[9] clearly demonstrated the predictive ability of MRI in regards to kyphosis progression after thoracolumbar spine fractures. More importantly, they also documented a distinct correlation between progression of post-traumatic kyphosis and poor functional outcome. Post-traumatic kyphosis was fairly well tolerated in the 1940s and 1950s by vigorous patients who had no spinal cord injury. Unfortunately, its existence provided a large group of symptomatic patients during the 1960s and 1970s. This group benefited both from correction of their spinal deformity, as well as decompression from deformity-induced spinal cord stenosis. This problem is rarely seen now in the United States but still is commonly seen in countries where "early surgical stabilization" is not regularly practiced.

Because randomized controlled trials of surgical versus nonsurgical management have not adequately been performed, strong opinions (of treating physicians and surgeons on both sides of the argument) characterize the recommendations made to injured patients. Both approaches have obvious risks and benefits. However, clinical experience of the senior author strongly suggests that earlier surgical stabilization of more severely displaced and comminuted injuries has—at least in his practice—almost eliminated the problem of post-traumatic kyphosis.

OPTIMAL CURRENT MANAGEMENT OF SPINAL FRACTURES AND FRACTURE DISLOCATIONS, WITH OR WITHOUT SPINAL CORD INJURY

The current state of the art management of spinal fractures and spinal cord injury in the United States includes both restoration of spinal alignment by either operative or nonoperative means, as well as direct or indirect decompression of the injured spinal cord and nerve roots to facilitate their recovery. The choice of optimal treatment depends to a great degree on the experience and judgment of the surgeon attending the patient. However, the basic principles are straightforward. Initial goals include reduction and maintenance of reduction until healing of the spinal fracture in "acceptable" alignment occurs. In the United States, this generally means surgical stabilization or immobilization in a brace. However,

in countries where nonsurgical management is preferred, prolonged bed rest in postural reduction with "acceptable alignment" may be the treatment of choice.

Laminectomy is avoided as an isolated operative treatment. However, in combination with open reduction of the spinal fracture and surgical stabilization with spinal implants, laminectomy certainly is useful. Laminectomy permits identification of the dural tube, provides a good source of autologous bone graft to facilitate spinal fusion, and permits the

surgeon to directly palpate and visualize the spinal canal as the reduction of the fracture is performed. In addition, laminectomy facilitates posterolateral decompression of the compromised spinal cord in necessary instances.

If open reduction and internal fixation is chosen, and the patient is young and active, a short-segment approach is generally selected. This involves the instrumentation of one healthy vertebra above and one vertebra below the fracture site (Fig. 27-4). If the patient has a psychiatric or drug-
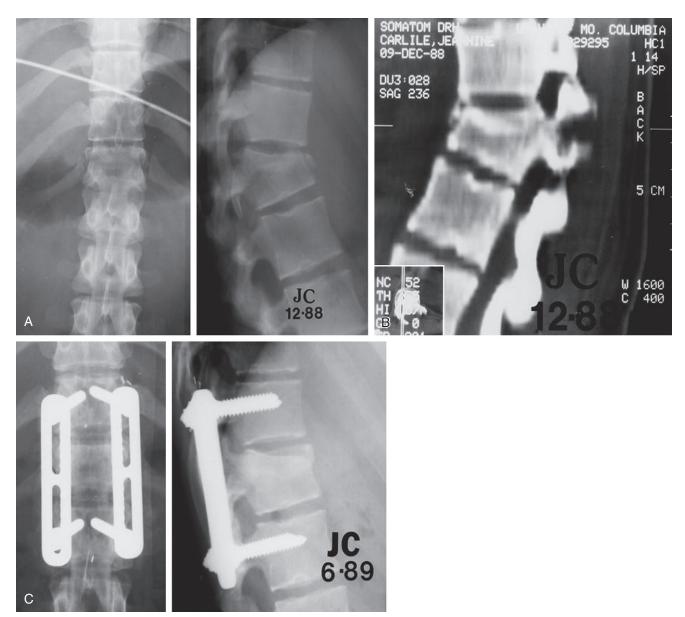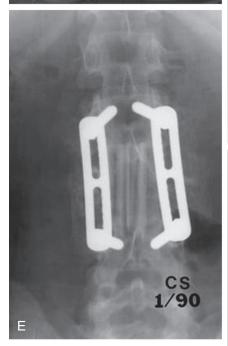

Fig. 27-4 *A,* Typical short-segment reconstruction of flexion-distraction injury via a posterior approach for a low point–total injury. Scoring as follows: involves only the top half of the vertebral body (2 points), minimal fragment displacement on the computed tomography (CT) scan (1 point), and more than 10-degree correction of kyphosis to restore normal sagittal plane alignment at T12 (3 points). Thus, the point total is 6 points. *B,* Axial CT scan and sagittal reconstruction are required in assessing point totals. *C,* The plain radiographs 7 months after surgery show short-segment reconstruction and healing in normal alignment. (From Parker JW, Lane JR, Karaikovic EE, Gaines RW: Successful short-segment instrumentation and fusion for thoracolumbar spine fractures. Spine 25:1157–1169, 2000.)
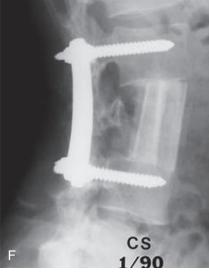
Fig. 27-5 *A, B,* and *C,* L3 severe fracture-dislocation involves the entire body (3 points), has displacement of fragments wider than 2 mm over the entire body (3 points) but requires only 4 to 9 degrees of correction to restore the sagittal plane (2 points). Translational displacement defines this injury as a fracture-dislocation, although the displacement is only moderate. *D,* Initial treatment involves posterior instrumentation, posterolateral decompression with suture of dural laceration and containment of displaced roots, and pnm osterolateral fusion with an autologous iliac crest bone graft. *E-F* Second-stage fibular anterior strut autologous graft is carried out 1 week later to support the posterior construct mechanically. Full neurologic recovery and fracture healing is obvious 8 months after surgery. (From Parker JW, Lane JR, Karaikovic EE, Gaines RW: Successful short-segment instrumentation and fusion for thoracolumbar spine fractures. Spine 25: 1157–1169, 2000.)
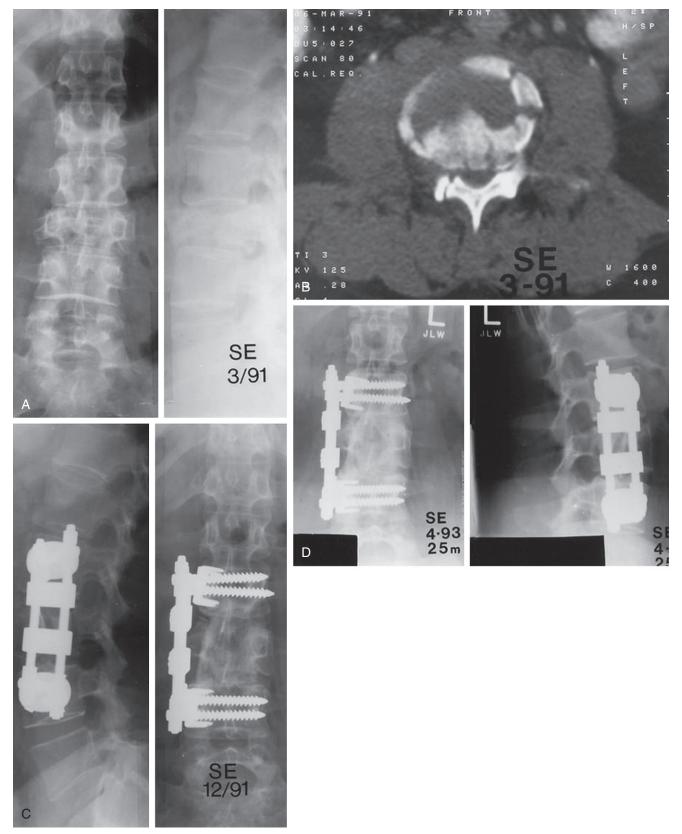
Fig. 27-6 *A,* A 21-year-old woman with total body involvement (3 points) with widespread fragments on CT *(B)* (3 points) that needs more than 10-degree sagittal plane correction because the injury is at L3 (3 points). The point total is 9. Thus, the patient is treated surgically from the anterior approach with Kaneda device and strut graft. Proper healing with no loss of reduction is illustrated at 9-month *(C)* and 25-month follow-up radiographs *(D).* (From Parker JW, Lane JR, Karaikovic EE, Gaines RW: Successful short-segment instrumentation and fusion for thoraco-lumbar spine fractures. Spine 25:1157–1169, 2000.)

abusing background, or other health problems that limit his or her ability to participate in postsurgical spinal bracing, then a long-segment approach can be used. This involves fixation of two, or even three, normal vertebrae above and below the fracture site to achieve stability. This long-segment approach may also be useful in the older patient with osteoporotic bone.

If spinal implants are used, and an open reduction performed, most surgeons simultaneously perform a spinal fusion over the instrumented segments. However, there are still some surgeons in Europe who prefer to instrument the spinal fracture without fusion and subsequently remove the implants a matter of months following the open reduction.

APPROACH FOR OPEN REDUCTION, SPINAL INSTRUMENTATION AND FUSION

It is difficult to reduce fractures with translational displacement from an anterior approach alone. Therefore, injuries with translation are often approached first posteriorly, to obtain reduction. Either a short- or long-segment approach is chosen. Ultimate choice of fixation depends on the aforementioned parameters as well as any coexisting injuries.

Thoracolumbar fractures with translation and severe comminution of the injured vertebral body require both posterior open reduction and instrumented fusion, followed by anterior augmentation. This consists of anterior partial vertebrectomy with cage or strut graft to provide reliable short-segment reconstruction (Fig. 27-5).

Highly-comminuted burst fractures without translation, which are particularly common at the thoracolumbar junction, can be nicely repaired from an anterior approach. Vertebrectomy and cage or strut graft reconstruction with anterior implants is then performed (Fig. 27-6). Again, the choice of short-or long-segment approach depends on the reliability of the patient and any coexisting injuries. Most

"low point total" injuries in the load-sharing classification can be successfully treated by the short-segment posterior approach, with pedicle screws.[3] If there is extensive ligamentous injury, involving either disk disruption with tissue in the spinal canal or extensive bony comminution of the injured vertebra, then short-segment posterior fixation requires anterior augmentation. Posterior instrumentation and fusion in these instances is often followed by anterior diskectomy, with or without vertebrectomy and strut graft or cage reconstruction. Postoperative immobilization of repaired injuries is generally recommended for most operatively stabilized injuries for 4 to 12 weeks, depending on the patient and his or her associated injuries.

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Bracing for Thoracolumbar Trauma

INTRODUCTION

The recent literature, including the randomized trial reported by Wood et al.[1] in 2003, confirms the central role of bracing and nonoperative management for the majority of thoracolumbar spine injuries. In this chapter, the terms *brace* and *orthosis* are used interchangeably. According to *Merriam Webster's Medical Dictionary*,[2] these terms refer to orthopaedic appliances used to support, align, prevent, and/or correct deformities or to improve the function of movable parts of the body. Nachemson[3] defined braces as external devices that "apply indirect forces to the spine for correction or prevention of deformity, stabilization, unloading, or supportive effects."

Spinal bracing commonly is prescribed for patients with deformity and degenerative disease. In 1987, more than 250,000 corsets were prescribed in the United States alone, most for low back pain.[4] In this chapter, we examine bracing for traumatic thoracolumbar spine lesions resulting from motor vehicle accidents, falls, acts of violence, and recreational sports. Trauma affects all spinal levels, from the occipitocervical to the sacrococcygeal junctions, with the thoracolumbar region being the most frequently injured.[5,6] These injuries affect both genders and all ages and races, but peak incidence is bimodal. High-energy injuries resulting from motor vehicle accidents and falls from great heights occur most frequently in male young adults. The second, older group typically presents with thoracolumbar trauma sustained during lower energy accidents, such as falls from standing height.[7–9] Treatment strategies in general, and bracing in particular, must consider the energy imparted to the injured tissues and medical comorbidities, including osteopenia and pulmonary disorders.

Descriptions of treatment administered to patients sustaining thoracolumbar spine trauma date to the Edwin Smith Papyrus from the 17th century BCE.[10] Then, as now, bracing had a central role in spine injury management. Other clear descriptions of early braces can be traced to Galen (131−201 AD).[11] Ancient Egyptian, Greek, and Roman orthoses were constructed of whale bone and tree bark but were not as crude as might be expected. Lumbar supports fashioned from tree bark and recovered by archaeologists from the cliff dwellings of the pre-Columbian Indians are reportedly similar to the thermoplastic spinal braces used today.

In Europe, by the 18th and 19th centuries, a variety of different braces fabricated from combinations of leather, steel, and plaster of Paris were being manufactured.[12] Those designs remained relatively static until the 1960s and 1970s when aluminum and thermoplastics replaced the heavier steel and leather designs. Regardless of the materials used, however, the indications for and usage of orthoses have not changed.

In patients with thoracolumbar trauma, orthoses currently are used in three settings: (1) as a stand alone as part of a mobilization program, (2) as part of a recumbency program, and (3) as an adjunct to surgical stabilization. In each setting, the brace can be designed to control pain, limit spinal motion, and/or protect the spinal column from additional loads that might otherwise cause progression of deformity (Table 28-1). In this chapter, we emphasize bracing as the chief element in nonoperative spine fracture management. Patients with thoracolumbar trauma who are treated with recumbency in rotating beds do not accrue much additional benefit from bracing.[13] Braces commonly are used as adjuncts to surgical stabilization. Temporary bracing might be used in patients for whom the indicated surgical stabilization occasionally is delayed for medical reasons. Postoperatively, braces might be prescribed to provide additional protection or as a kinesthetic reminder. Such adjuvant bracing is particularly appropriate in osteoporotic patients whose construct rigidity might be compromised.[14] In patients with normal bone, the surgeon might choose to limit gross spinal motion until postoperative muscle strength and spasm have improved.

In short, some thoracolumbar injuries are more likely than other injuries to successfully heal in a brace. Selection of the proper brace and correctly instructing the patient in its

TABLE **28-1**   Brace Functions in Thoracolumbar Trauma*

Pain control
External stabilizer of the spinal column
Mechanical unloading of spinal column
Range of motion/kinesthetic restriction/reminder
Postural reminder
*These functions apply whether the brace is used alone or as an adjunct to surgical management.

use will significantly improve the likelihood of a satisfactory outcome. In this chapter, we seek to provide enough information to allow the physician to devise successful bracing strategies for a variety of thoracolumbar injuries.

BIOMECHANICS OF SPINAL TRAUMA AND NATURAL HISTORY OF HEALING

The generic prescription to "brace" a trauma patient is more likely to fail if the treating physician does not understand the mechanics and biology of the injury and healing processes. Moreover, successful bracing strategies require an understanding of brace function, in particular its abilities to provide stability and to counteract the vector of force induced by the injury itself.[15]

A number of classification schemes has described the wide range of appliances that are charged with the tasks of limiting spine motion and improving or maintaining alignment. Generally speaking, in terms of decreasing rigidity, they are spinal fixators, casts, conventional orthopaedic appliances, and corsets. Fixators, such as the halo vest, connect directly to cortical bone and are not used to treat thoracolumbar trauma.

Conventional orthopaedic appliances require direct contact with the skin and soft tissues of the head, neck, thorax, abdomen, or pelvis. Classification of these devices can be further divided into limited contact and extensive coverage categories. Limited contact braces are comprised of discrete pads and straps (as with the Jewett brace). Extensive (or total) coverage devices are best represented by the full contact orthosis (or thoracolumbosacral orthoses [TLSO]). Considering that body casts are mechanically similar to total contact braces, the two are considered together.

Historically, prescriptions for spinal braces used myriad confusing eponymous names and contradictory descriptors. A task force of orthotists, spine surgeons, and other health officials created the Harris Classification as a common, consensus nomenclature for conventional spinal orthoses.[16] In general, this system names orthoses based on the regions they span. Thus, a cervical orthosis covers only the neck whereas a cervicothoracolumbosacral orthosis spans the entire spine. A broad variety of cervical orthoses of interest to the spinal

traumatologist is available. Use of the longest of these braces, such as the halo cast, halo vest, and Yale brace, might be indicated in some upper thoracic injuries.[17] In particular, most four-poster and sternal occipital mandibular immobilizer designs afford little control at the cervicothoracic junction. Yale and Guilford braces afford some control at the cervicothoracic junction but rapidly lose effectiveness below T2. Typically, upper thoracic injuries are treated by adding a cervical extension to a thoracolumbar orthosis.

Within in a particular region, braces might be subdivided into full (e.g., TLSO) and limited contact (e.g., Jewett) braces. Molded, full contact braces offer more control of lateral bending and rotation, particularly at the thoracolumbar junction. Most limited contact braces are useful mainly for flexion and extension control in more stable injuries, such as compression fractures.[18] In the finite element modeling of compression injuries presented by Patwardhan et al.,[19] residual spinal stiffness was at least 50% of normal. Jewett bracing effectively prevented deformity under physiologic flexion loading. This brace was ineffective for two-column injuries that reduced stiffness to between 50% and 85% of normal.

BRACE MECHANICS

Spinal bracing is principally similar to long bone casting. Rigid immobilization requires multiple motion segments above and below the injured level. Historically, the axial skeleton is divided into five segments: the skull, the cervical, thoracic, and lumbar spine, and the sacrum-pelvis. Using these divisions, rigid (i.e., two-level) spinal immobilization is available only in the thoracic spine (with a halo attachment, TLSO, and hip spica).[20] More typically, semirigid immobilization is sought. Ideally, the brace extends the same distance cranially and caudally from the injury. Benzel[20] recommends extension for four or five vertebral levels. More unstable injuries require longer braces.

Although internal fixation applies forces, spinal column orthoses apply force from a distance. The soft tissue thickness separating the spine from the inner surface of the brace is inversely proportional to the resulting effectiveness of immobilization. On the other hand, longer braces provide more stability than shorter ones because they have longer soft tissue contact intervals. These concepts and brace efficacy can be summarized by a length-to-width ratio (Table 28-2).

TABLE **28-2**   Mechanisms of Brace Function

Direct skeletal fixation
Off-loading or axial traction (less effective in thoracolumbar spine)
Three-point bending
Increase in functional diameter of spinal column
Fluid compression
Kinesthetic reminder

Fig. 28-1 Clinical photo of a healing sacral decubitus noted in a L1 burst fracture patient managed in a brace. His ambulatory status was markedly limited by bilateral calcaneus fractures.

Spinal immobilization improves with greater conformation of the brace to the body and rigidity of the tissue against which the brace anchors.[21] Rigid structures (skull, ribs, and pelvis) provide greater holding power than more liquid body elements (abdomen). Excess adipose tissue between the brace and its anchoring structure requires longer braces to achieve the same immobilization.

Internal fixation is limited by failure at the screw-bone interface. Orthoses are limited by the much lower forces the skin can tolerate (Fig. 28-1). Variability in forces generated by the brace might relate to how tightly the brace is worn. Casting eliminates concerns regarding brace compliance and uniformity of application (such as tightness); however, it is more difficult to inspect the skin for pressure sores with casts. Moreover, the patient's body will react to the constant pressure of the cast by redistributing subcutaneous fat and fluid. These shifts also diminish the forces generated by the cast.

INJURY MECHANICS BY LEVEL AND INJURY PATTERN

The inherent stability of the spine, its susceptibility to various patterns of injury, and the degree of stabilization a brace might offer vary by craniocaudal level. The thoracic spine enjoys an inherent splinting effect provided by the rib cage; however, the energy-absorbing effects of fat, muscle, and breast tissue again limit the ability of a brace to provide or maintain distraction.[22] Less mobile than the cervical or lumbar regions, the thoracic spine exhibits greater rotation than flexion. More caudally, lateral bending increases while axial rotation decreases.

In the upper thoracic spine, however, the benefits of thoracic cage rigidity are diminished by the mobility of the shoulder girdle. This mobility and the shoulder's thick covering of muscle and subcutaneous fat limits the ability of shoulder straps to maintain consistent distraction.[22] Changes in shoulder position and alignment from supine to standing further diminish the impact of distractive braces. Bracing for injuries above T7 usually requires cervical extensions (Fig. 28-2).[23]

At the thoracolumbar junction, the transition from thoracic kyphosis to lumbar lordosis accompanies a change from coronal thoracic facet orientation to the sagittal lumbar facet joints. Absence of a lordotic or kyphotic curvature subjects the levels between T10 and L2 to increased risk of axial loading injury (burst injury). Further, this segment also lacks protection from the rib cage or pelvis. On the other hand, bracing of thoracolumbar junction injuries benefits from the long truncal length above and below.

Although burst fractures also occur in the lower lumbar spine, sagittal facet orientation and lordotic contour results in different injury mechanics.[24] For example, kyphotic collapse is less common. Both full and limited contact braces poorly control low lumbar and sacral segments because distal purchase over the sacrum is poor.[25] Minimal lumbar rotation with high flexion and extension increases erector spinae activity when the patient wearing a brace ambulates. To improve TLSO or lumbosacral orthosis (LSO) effectiveness in the management of injuries between L3 and S1, a hip spica attachment is added.

The thigh piece can be placed on either side, typically the more symptomatic side, and adds approximately $500 to the cost. The cuff extends to 5 cm above the patella. Maximal effectiveness requires marked reduction of pelvic inclination.[26] Clinically, acceptance of thigh spicas is limited because of the marked effect on walking, sitting, and transfers. Some patients will require a cane for ambulation when in the brace. For many older or polytraumatized patients, donning and doffing these braces is impractical in a home setting and requires placement in a rehabilitation unit or nursing home. To compromise, the hip often is held in 20 degrees of flexion to allow sitting and walking. At some point during the healing process, the thigh spica can be removed to improve ambulation and rehabilitation potential. Fidler and Plasmans[27] found that this type of thigh spica brace reduces gross pelvic motion more than a conventional TLSO, but segmental translation with axial loading was not affected.[26,27]

After spinal level has been considered, bracing strategies are based on the degree to which the injury destabilizes the motion segment. Lower energy injuries, such as lumbar strains and mild compression fractures, only minimally disrupt stability and do not "require" bracing. In such cases, a corset might improve pain control. In patients with poor underlying tissue quality, however, minor injuries might markedly destabilize the spine (e.g., ankylosing spondylitis).

Moderate energy injuries include more severe compression fractures and some burst and Chance fractures. Injuries in this group benefit from stabilization with either surgical

fixation or external immobilization. The most severe injuries, such as fracture dislocations, are so disruptive that bracing will not adequately restore stability. These high-energy injuries require surgical stabilization.

The residual spinal stability associated with various common injury patterns can be compared. For example, Ching et al.[28] re-created three common injury patterns in 26 cadavers. Increased spinal laxity and decreased residual stability were characterized by an increased neutral zone. Compression fractures retained the greatest stability, with only a 56% increase in flexion and extension. Burst fractures and flexion-distraction injuries were similarly unstable with increases in the neutral zone in all planes. With

burst fractures, the flexion and extension neutral zone increased 154%.[28]

In general, flexion-compression and axial-loading injuries are more amenable to bracing than are translational or torsional injuries. Which burst fractures are "too unstable" for brace management remains highly controversial. In lower energy burst fractures, the posterior neural arch remains intact. With further vertebral collapse and wider fragment displacement, the pedicles splay, resulting in a vertical laminar split. Cumulative involvement of the anterior, middle, and posterior columns yields progressive mechanical destabilization. Once the posterior column fractures or the posterior ligaments tear, maximal
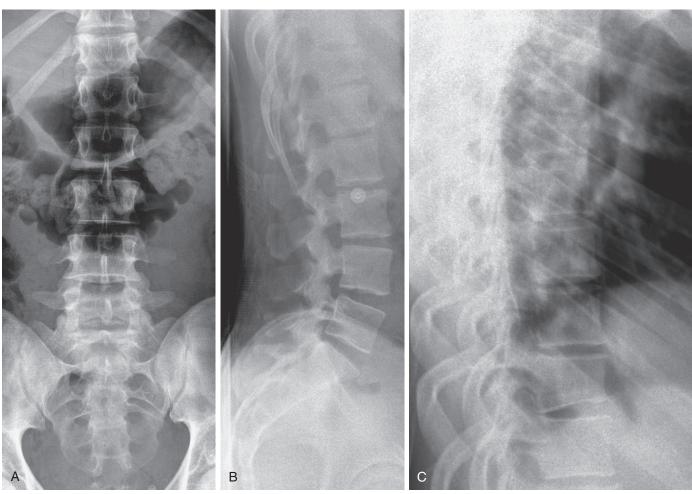

Fig. 28-2 This 21-year-old college student presented to the emergency room on transfer from a hospital in Colorado where he had been taken after a snowboarding accident which included a 20 foot fall onto his back and buttocks. He had no prior trauma history. He had diffuse back pain, but no myelopathic or radicular complaints. Initial plain films were suggestive of multiple compression deformities *(A to C)*. A thoracolumbar computed tomographic scan was ordered and demonstrated 4 levels of compression fracture and mild burst fracture *(D and E)*. Representative axial views *(F and G)* fail to demonstrate significant comminution, canal compromise, or marked disruption of the anterior weight-bearing column, so bracing was recommended. Initially, the patient was placed in a TLSO *(H and I)*. By 12-week follow-up, the patient was pain free and had healed in radiographically acceptable alignment *(J to L)*. Note the relative hypokyphosis of the mid-thoracic spine with the increased kyphosis of the upper thoracic spine. Given the high thoracic fracture, a cervical extension to the TLSO could have been considered.
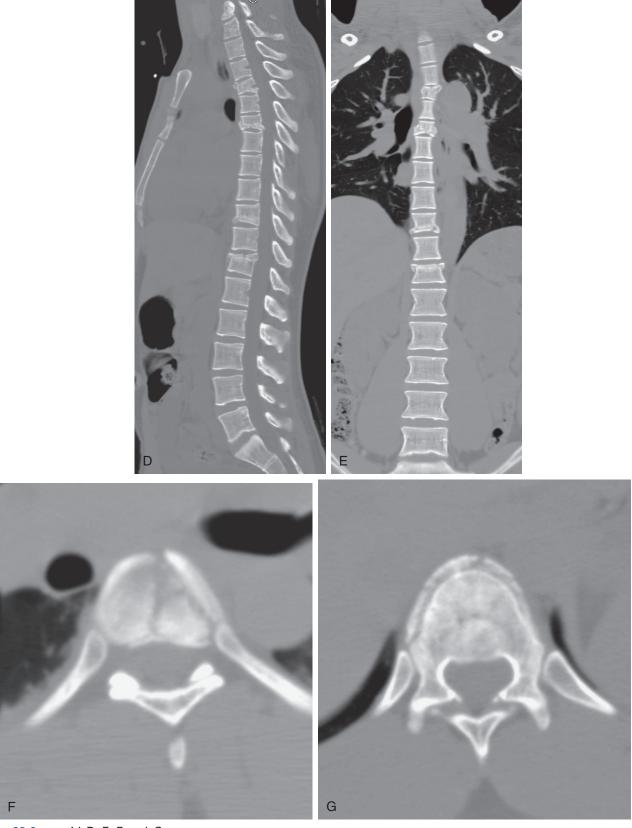
Fig. 28-2, cont'd *D, E, F,* and *G*

Fig. 28-2, cont'd *H, I, J, K,* and *L*

mechanical instability results and the likelihood of successful brace management decreases.[29]

BRACING AND FRACTURE HEALING

Healing of injuries to the bony elements is more likely to restore mechanical competence of the spinal column than are ligaments ruptures. The time course of this mechanical restoration varies widely. The fracture remodels for at least 1 year.[30,31] The effects of age, osteoporosis, and pharmaceuticals such as anti-inflammatories and bisphosphonates on healing time, remain controversial.

During the healing interval, spinal deformity might increase. In children younger than 6 years, gradual improvements in postfracture vertebral deformity and sagittal balance can continue for decades. On the other hand, spinal fractures might also injure the growth plates in young patients, precipitating relentless post-traumatic scoliosis or kyphosis.[32]

The most important functions of bracing involve augmentation of the motion segment's residual mechanical stability. This function can be achieved through one or several means. First, as with casting of the appendicular skeleton, limitation of motion accelerates healing.[33] The degree to which bracing improves healing and the differential effect of more or less restrictive bracing is not clear.

Second, braces might decrease spinal loading. In the cervical spine, distraction can be achieved, but it is more difficult in ambulatory patients with thoracolumbar trauma. The brace might also limit loading by signaling the patient's friends and family to avoid exuberant contact or by acting as a reminder to the patient to avoid dangerous activities. Most important in the selection of an appropriate brace for a particular injury, however, is an understanding of the appliance's ability to restrict motion.

All braces limit gross trunk movement to some degree, and whole spine motion restriction is therefore a universal mechanical rationale. Different braces limit gross trunk motion to different degrees and might not necessarily limit segmental motion at all. Improperly fitted braces might limit gross spinal motion but increase segmental motion at the injured level.

Orthoses able to reduce segmental motion, on the other hand, are assumed to also reduce overall spinal motion. Segmental stabilization involves diverse mechanisms, such as three-point bending, increased spinal cross-sectional area, and active control. As a metaphor for these mechanisms, Woodard et al.[22] described the spine as an ideal column with a fixed base and free upper end. With initial loading, the spine shortens. Excessive load causes the column to buckle. The magnitude of this load at failure is proportional to column's modulus of elasticity multiplied by the square of its cross-sectional area over the square of its length. The ability of the spine to bear higher loads without buckling can be improved by increasing its cross-sectional area or decreasing its length or elasticity.

Almost all thoracolumbar orthoses use three-point bending principles and apply balanced transverse forces to resist bending forces and contribute to axial load bearing.[34] Highly conforming sleeve orthoses, such as clamshell TLSO, rely less on this principle than do limited contact braces, which often apply dorsal loads to the upper thorax and pelvis and ventral forces at the midportion of the brace. Physically, these limited contact braces are diverse but usually consist of two end-fixation elements and a connecting, longitudinal member. For example, a chairback brace purchases the rib cage with a thoracic band and the hips with a pelvic band. Uprights connect the thoracic and pelvic bands. The Jewett brace applies a dorsal force at the sternum and pubis. Then, the posterior band is positioned at the injured segment to direct a ventral force.[35,36] Longer lever arms reduce the force required to produce sufficient bending moments. Three-point bending mechanics effectively divide the spinal column into two shorter lengths, thereby increasing the load to failure of the whole column.[22]

The bracing principle of fluid compression relies on a functional increase in the cross-sectional area of the spine. Circumferential compression of a soft, fluid-filled abdomen creates a semi-rigid fluid cylinder.[3] A fluid cylinder is mechanically incompressible and thus able to share some of the vertebral body's axial load, increasing its effective cross-sectional area. Because of airway issues, fluid compression effects are not useful in the cervical spine but are a mainstay of lumbar corsets and similar short-length, sleeve orthoses. In patients with back pain, corsets reduce intradiskal pressure but do not measurably increase intra-abdominal pressure.[37] The fluid column is anterior to the spine, and these braces therefore mainly limit sagittal plane motion.

The next segmental motion control mechanism, transverse loading, is not commonly used in thoracolumbar bracing. Balanced transverse loading seeks to restrict spinal rotation and translation. Effective bracing requires strict appliance centering at the axis of rotation of the injured level. Long arms projected transversely out of the braces create the necessary moment arm. Moreover, most translational and rotational injuries are ligamentous and, even if motion could be controlled, healing remains unreliable.

Active control uses the normal contributions of muscular force to spinal stability as a mechanical principle in some thoracolumbar braces. Absent the normal opposing muscular forces acting on the spine, small 2-kg loads cause the spinal column to buckle.[22,38] The opposing actions of the flexor and extensor combine to stiffen the spine and increase its load-bearing capacity through increases in the effective cross-sectional area and modulus of elasticity.[21] Clinically, this phenomenon is observed in patients with spinal fractures who report a sense of stability for the first 30 to 60 minutes out of the brace. During that interval, the intrinsic, muscular mechanisms for spinal stability allow the patient to maintain alignment and to off-load the injured segment. Over time,

however, these muscles fatigue, and without bracing, the injured segment becomes overloaded.

For patients with excellent core strength, muscular control alone might provide adequate immobilization of less unstable injuries. Older, osteoporotic, or polytraumatized patients rarely have enough muscular strength to effectively use this principle.

Some braces are intended to improve native dynamic control mechanisms. Many orthoses promote muscular activation through tactile feedback. Pressure at the brace-skin contact site reminds the patient to maintain a preferred spinal alignment position and to limit unsafe, gross body motions.[25] In that setting, only intrinsic muscular support limits undesirable motions but the muscles are guided by the brace. Stiffer braces worn securely are more insistent with proprioceptive reminders than are softer braces. On the other hand, this uncomfortable "feedback" limits brace compliance.[35] Moreover, more rigid orthoses lead to greater degrees of extensor atrophy and loss of vital capacity.[19] A recent randomized trial of a novel brace design, the Spino-med brace (Medi-Bayreuth, Bayreuth, Germany), revealed a 73% increase in extensor strength, 58% increase in abdominal flexor strength, 11% decrease in kyphosis angle, and 58% increase in abdominal flexor strength in half of the 62 enrolled patients receiving the brace (Fig. 28-3).[39]

SURVEY OF BRACES

TLSO AND RISSER CASTS

Today, the majority of high-energy thoracolumbar fractures are treated with the use of TLSO. Modern TLSO are made from polypropylene, which is strong enough to allow a relatively thin and conforming brace to fit under clothing. In adult sizes, the devices weigh 3 to 4 pounds and cost between $1200 and $2220.

Historically, patients with thoracolumbar fractures have been positioned on a Risser hyperextension table and have undergone casting.[40] The casts are heavy and limit access to the trunk.[41] Such limited access adversely affects hygiene and might also preclude early detection of skin breakdown. Casting should be avoided in patients at higher risk of intra-abdominal injury, such as extended ileus, abdominal wall contusion, and lap belt injury. Although Risser tables are still the de facto form of spinal immobilization in many prestigious hospitals around the world, most hospitals no longer have them. For thinner patients, a Jackson spine surgery frame can be substituted. Hyperextension and casting undoubtedly improve reduction, but most outcomes reports show clear reversion to the initial postfracture alignment during follow-up.[1,33,42] Today, casting is most often used for children, uncooperative patients, and patients with traumatic brain injury.

Three common methods to prescribe a TLSO are off the shelf with or without a reduction maneuver, custom-molded with or without a reduction maneuver, and conversion to
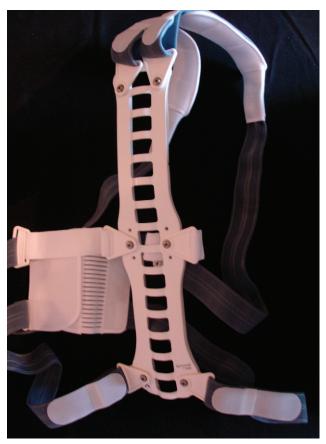

Fig. 28-3 Spino-med brace for osteoporotic compression fractures.

TLSO after a short period of extension casting. A well-made TLSO or body cast uses the principle of fluid compression to increase abdominal intracavitary pressure, providing another force transmission column and off-loading the bony spinal column. Efficient force transmission is provided by distributing this pressure over a wide surface area. Like most braces, the TLSO best limits flexion and extension and then side bending. A well-fitted brace will control rotation and translation but only to a limited degree. A stereophotogrammetric analysis of seven patients in both LSO and molded TLSO braces showed that neither device significantly restricted translation in the sagittal, vertical, or transverse planes (Fig. 28-4).[25]

General indications for TLSO immobilization include the following:

1. Immobilization of moderate- to high-energy flexion-compression or axial-loading injuries
2. Immobilization of multiple low-energy fractures, such as osteoporotic vertical compression fractures (VCF). TLSO use for this patient group is particularly helpful if collapse occurs during early follow-up or if the patient is not a candidate for a limited contact brace.
3. Although a standard, custom, or off-the-shelf TLSO can be used for patients with idiopathic scoliosis, special full-contact scoliosis orthoses, such as the Boston
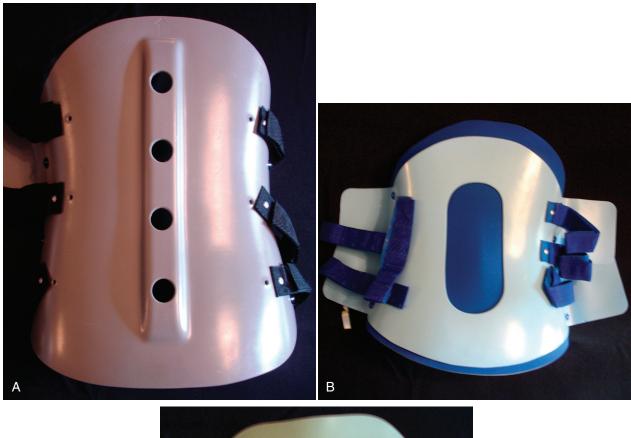
Fig. 28-4 This off-the-shelf TLSO system includes two options for its posterior panel: the standard straight and closed back *(A)* and a back portion with a central cut-out *(B)* for postoperative patients. The bracing system is gender specific, also, and this anterior panel *(C)* includes cut-outs for the breasts.

overlap brace, are also commonly used. Because those braces have little role in trauma, they are not further discussed in this chapter.

4. Immobilization of other destabilizing spinal disorders, such as tumors and infections

A well-fitted TLSO provides stabilization from T3 to L3 and a radiographically measurable improvement in the sagittal balance of the spine across the treated segments. Toward the limits of the stabilization zone of the TLSO cranially or caudally, modifications improve. Cervical and hip spica attachments were described previously. The superior trim line can be extended on the anterior shell to the deltopectoral groove to decrease more cranial flexion moments but will also limit upper extremity reach. Sternal extensions and back supports allow ready interconversion from TLSO to LSO. With some braces, even the flexion and extension stiffness can be "tuned down" to allow a gradual return of extensor muscle strength without completely compromising protection.

Other brace additions improve patient comfort and compliance. Although off-the-shelf braces typically are less expensive and can be made available to the patient sooner after trauma, by their very nature, they are not as effective in matching the individual body contours of the patient. On the other hand, brace manufacturers now routinely offer off-the-shelf braces with gender-specific liners and, occasionally, a few options for different body types. These adjustments allow the brace to more closely follow body contour variations from torso to waist and hips. Similarly, inserts can be added to adjust lordosis from 0 to 40 degrees to improve both comfort and control. Along with straps to adjust control by body position, these liners can redistribute pressure away from the ilium, trochanters, and ribs, which in turn decreases brace-associated discomfort and increases compliance. Along the same lines, some braces have open centers or flat back designs that might be appropriate for postoperative patients.

The literature comparing different brands and configurations of TLSO is limited. In 2003, Cholewicki et al.[43] studied three TLSO brands for differences in motion restriction and comfort. The authors measured spinal motion restriction and passive trunk stiffness provided by the CAMP and Aspen TLSO and the Boston Body Jacket and found no significant measured differences. The study participants did not perceive differences in restriction of motion from one brace to another either but found the Aspen TLSO to be more comfortable than the other two orthoses.[43]

LIMITED-CONTACT ORTHOSES

Although many limited-contact thoracolumbar orthoses are available, the most common varieties are Jewett and cruciform anterior spinal hyperextension (CASH) braces. These lightweight braces with open brace designs have some advantages, but the force applied over a small surface area to the sternum and the pubis might cause enough discomfort to limit compliance.[20] Hyperkyphotic patients are more likely to complain (Figs. 28-5 and 28-6).

The Jewett hyperextension brace uses a three-point pressure system with one posterior and two anterior pads. The anterior pads confer posterior pressure to the sternum and pubic symphysis. The posterior pad crosses the mid-thoracic region at the point at which its anteriorly directed pressure opposes the anterior pads to extend the spine. The Jewett's lightweight design can be adjusted from a lateral axillary bar to which the anterior pads attach. The brace tends to "ride up" when the patient is seated, so it should be fitted in that more demanding position. When seated, the sternal pad should run a half-inch inferior to the sternal notch and the pubic pad crosses a half-inch superior to pubic symphysis.

The Jewett hyperextension brace limits flexion and extension between T6 and L2-L3.[19,44] It does not limit lateral bending or rotation. Jewett braces cost approximately $500 and typically are indicated for compression fractures of the lower thoracic spine, although some authors report successful use in cases of higher energy trauma.[45] Contraindications include injuries above T6, where the sternal pad increases segmental motion. The Jewett brace is one of the most commonly prescribed for osteoporotic VCF, but some authors prefer more cylindrical immobilization. This brace does not provide abdominal support, and some patients have used it in conjunction with an abdominal binder. The Korsain brace represents a Jewett modification with "built-in" abdominal support.

The CASH brace, like the Jewett, is composed of anterior sternal and pubic pads that produce posterior forces opposed by a posterior pad and strap. The sternal and pelvic pads attach anteriorly to a cross-shaped metal bar that can be bent
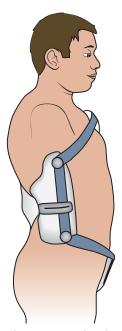

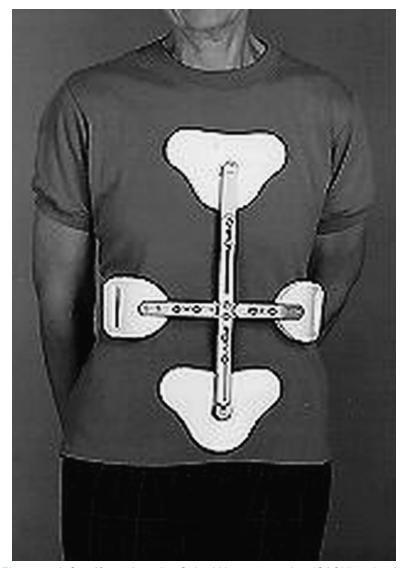Fig. 28-5 A Jewett hyperextension brace.

Fig. 28-6 A Cruciform Anterior Spinal Hyperextension (CASH) orthosis.

to adjust chest and pelvis pressure. A CASH brace costs approximately $500 and has indications and contraindications similar to those for the Jewett brace.

The CASH brace is increasingly prescribed for patients with lower energy vertebral compression fractures. This brace is lighter, more cosmetically appealing, and, for some patients, more comfortable than the Jewett brace. The CASH brace provides greater breast and axillary pressure relief than does the Jewett brace, and two round upper chest pads can be used instead of the sternal pad to decrease breast discomfort. We recommend keeping both braces in the office and allowing the patient to decide between them after trying them. Alternatively, the orthosis prescription might instruct the orthotist to fit the patient with whichever brace is most comfortable and effectively immobilized. The CASH brace is easy to don and doff but difficult to adjust once in place.

Whereas both Jewett and CASH orthoses function well in the sagittal plane, they do not decrease coronal and transverse plane motion. Overall, the Jewett brace probably provides more flexion and extension limitation than does the CASH brace. In recent years, a number of proprietary brace designs have been offered to improve comfort and compliance form patients with osteoporotic VCF. The effectiveness of the immobilization of these devices remains largely unstudied, but the devices can be considered for patients who refuse to wear the more standard Jewett or CASH style brace.

LSO

LSO attempt to stabilize the lumbar and sacral regions by encircling the upper abdomen, rib cage, and pelvis. Secure immobilization is limited by poor soft tissue purchase (especially in obese patients) and the inability to use three-point bend

mechanics. Fluid compression of the abdominal cavity is thought to have a role in stabilization, but experimental measurements of this effect have not been consistent.[21] As a result, although LSO frequently are prescribed for degenerative back pain, they are controversial for trauma patients (Fig. 28-7).

Semantic differences affect determining when a corset becomes an orthosis. Some authors list corsets as orthoses. Typically, however, an orthosis has at least one rigid segment. This group contains dozens of different brace types with various minor and major modifications but can be broken into three subtypes: corset with stays, chairback brace, and Knight brace.

Norton and Brown inserted wires into the spinous processes of volunteers to measure lumbar motion before and after bracing. They noted a paradoxical *increase* in lumbosacral motion while sitting in the brace. This increase was thought to be caused by stress concentration at the caudal portion of the supported segment.[36] Waters and Morris[46] used surface electromyography to monitor paraspinal muscle activity in and out of an LSO. The brace reduced muscle activity when standing but increased with fast walking, presumably because of altered pelvis rotation. Lantz and Schultz[47] measured trunk motion and surface myographical activity in a molded TLSO and various types of LSO. The TLSO was most effective in limiting motion. The LSO reduced flexion by 20% during flexion and extension and lateral bending and reduced rotation by 45%. LSO myoelectrical activity varied widely from a 9% reduction to a 44% increase.[47]

The standard, rigid LSO is comprised of plastic anterior and posterior shells that overlap for a tight fit. A Velcro closure anteriorly aids easy donning and doffing. Like a TLSO, which extends further along the thoracic cage, rigid LSO often are custom molded over the iliac crests for improved fit and can be trimmed to improve patient comfort or to offload injured areas. The brace might be used in the shower and is cleaned and dried easily. Motion restrictions include some limitation of flexion and extension with even less limitation of rotation and side bending. These braces cost between $500 and $1000 and are indicated mainly for midlumbar compression fractures or for postsurgical lumbar immobilization.

Most of the other standard LSO represent efforts to improve comfort by removing rigid brace elements where they are not thought to be necessary. For example, the Knight-Taylor brace incorporates a corset front with lateral and posterior uprights. Shoulder straps are said to further limit lateral bending and flexion and extension but might cause discomfort. The Knight-Taylor is typically off the shelf and prefabricated with polyvinyl chloride or aluminum. The posterior portion of the brace adds cross supports below the inferior angle of the scapula and a pelvic band fitted at the sacrococcygeal junction. The anterior, corset portion is made of laced canvas and is said to provide increased intracavitary pressure in the abdomen. The average cost of a Knight-Taylor brace is $750 and is indicated to limit flexion in patients with thoracic or lumbar compression fractures. As with most of these designs, flexion and extension limitations exceed those of lateral bending. Rotational control is poor.

The chairback brace consists of a rigid short LSO with two posterior metal or plastic uprights that run along the

Fig. 28-7 *A*, Modern, rigid off-the-shelf LSO with a closure mechanism that adjusts easily when the patient goes from sitting to standing and back. *B*, Lordosis can be adjusted with screws.

paraspinal muscles. Thoracic and pelvic bands improve the three-point pressure mechanics of the LSO and allow custom fit. An abdominal apron includes anterior straps to increase intracavitary pressure. The thoracic band runs 1 inch below the inferior angle of the scapula and extends laterally to the midaxillary line. The pelvic band is placed as caudally as possible without interfering with sitting comfort and typically is placed lateral to the mid-trochanteric line.

Chairback braces function best between L1 and L4, with a 45% limitation of lateral bending, reasonable limitations of flexion and extension, and minimal rotational control. This brace usually costs approximately $500 and occasionally is used in trauma patients for lower energy fractures of the midlumbar spine. The chairback is used more frequently for patients with back pain to unload the intervertebral disk and as a kinesthetic reminder after surgery. A slightly more expensive version of the chairback adds an "ortho-mold" rigid plastic back piece that is custom molded to the patient and inserted into the canvas.

The Williams Flexion Orthosis, a commonly prescribed LSO, is sometimes useful for patients with soft tissue strain. With this device, pelvic and thoracic bands are attached to oblique lateral stays with an elastic front. The anterior elastic apron allows flexion and therefore is contraindicated for patients with compression fractures or axial loading injuries. The brace effectively limits extension and could be considered for patients with spondylolysis.

The lightweight and comfortable corset with stays is one of the least immobilizing LSO. Anteriorly, this brace spans the area between the xiphoid and pubic symphysis. Posteriorly, the brace covers the lower scapula to the gluteal fold. Corsets function by increasing intracavitary pressure and are most effective in limiting flexion and extension. Stays are metal bars contained within the cloth or canvas of the corset that further decrease motion, most likely by functioning as a kinesthetic reminder. The stays can be custom molded and usually are placed posteriorly but can be placed in any position to limit motion in that direction. A corset with stays costs approximately $200 and is indicated for low-energy injuries without significant spinal column destabilization or as a step-down from other, more rigid devices. Similarly, the stays can be removed as the injury heals and less immobilization is required.

CORSETS

Corsets include a variety of devices made nearly entirely of cloth or some other, nonrigid material. As with soft cervical collars, they have little power to immobilize the spine and have limited usefulness in the management of acute thoracolumbar trauma. Examples include the sacroiliac orthosis, developed for pelvic instability after traumatic disruption or postpartum hyperlaxity, which, if effective at all, works as a reminder to maintain proper posture. These devices rarely are used today.

More common today are binders, which are cloth wraps used to limit regional motion. Thoracic binders are controversial today because of their negative impact on pulmonary mechanics, but they had been extensively used for symptomatic relief after rib fracture. Abdominal binders are used for support of weak abdominal musculature and might be of use in quadriplegics for whom the associated diaphragmatic support reduces pooling in the splanchnic circulation.

The most common member of the corset group is the lumbosacral corset. This device encompasses a range of devices, from those similar to binders (such as "lifting belts") to more rigid devices similar to true orthoses (such as corsets with metal stays). The short height and canvas construction of the typical corset allows it to do little more than reduce gross trunk motion. White and Panjabi[34] described a series of compression fractures with less than one-third anterior height loss. The authors thought the fractures were stable and recommended active exercise and mobilization after a period of bed rest to allow acute symptoms to subside. Although no orthosis was prescribed to improve stability, a lumbosacral corset was recommended to reduce trunk motion and thus pain.

CLINICAL EVALUATION

HISTORY

A clear understanding of the mechanism of injury will allow the examiner to "key in" to critical areas. For example, in patients who have sustained burst fractures from falls, assessing for calcaneus and other foot and ankle injuries is important. Occasionally, associated injuries preclude effective brace management. Sternum fractures, in disrupting the "fourth column" of the thoracic spine, can signal marked instability. Even if the spine retains adequate stability to successfully heal with brace management; however, sternum or thoracic cage pain adversely affects patient compliance.

The mechanism must also be compared with the severity of the spinal injury in question. For patients sustaining very high-energy trauma, the mild compression fracture observed on supine radiographs can actually represent a fracture dislocation. On the other hand, a patient sustaining a burst fracture after a simple fall from standing height might require investigation into bone loss states or neoplasm.

The relationship of pregnancy to thoracolumbar trauma has received little attention in the literature. In later pregnancy, spinal biomechanics change as the spine pivots around the enlarging fetus, decreasing compressive forces and increasing tension. As such, flexion-distraction injuries are more common in pregnant women than are the flexion-compression injuries that most frequently occur in the population at large.[48] If enough force is delivered to the spine of a pregnant woman, tension failure through the ligaments

occurs, precluding effective bracing. The body habitus in later pregnancy also limits bracing efficacy.

One should obtain a clear description of pain location and note whether the pain distribution has changed over time. Fracture and ligament injuries might be most painful in the midline, whereas belt-line low back pain might represent associated muscle spasm. One should inquire about radicular symptoms and the often missed bowel and bladder complaints or myelopathic symptoms such as ataxia.[49]

PHYSICAL EXAMINATION

Complete visual inspection of the patient is necessary. Aspects of body habitus, such as obesity and trunk length, are noted because they might affect decision making and brace selection. During follow-up, one should note changes in body weight as they affect brace fit.

One should document preexisting deformity and any change in coronal and sagittal spinal contour during the follow-up interval. Gait changes should be noted, as should abdominal distention or unequal chest expansion with deep inspiration. Close neurologic and musculoskeletal examinations are required. One should check for tenderness or gapping between the spinous processes, bruising, rib pain, and muscle spasm.[50]

Over time, spinous process tenderness and pain with flexion and loading subside, signaling fracture healing. Often, belt-line low back pain increases in braced patients. With restoration of boney stability, low back pain, spinal range of motion, and extensor weakness are treated with physical therapy. Similarly, braces can lead to loss of shoulder or hip joint range of motion. In less mobile patients, one should check for dorsal pedal edema or a Homan's sign. The skin under the brace should be inspected, particularly in insensate and hard to visualize areas along the back.
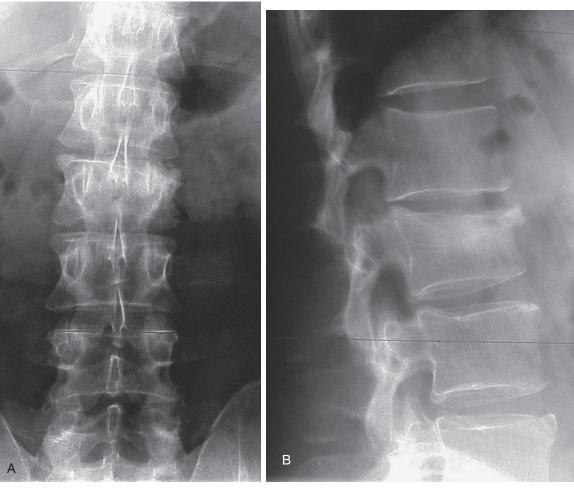

Fig. 28-8 This 42-year-old man with a history of moderate, intermittent back pain presented with significantly increased pain after a hockey injury. He was found to have an L2 vertebral compression fracture and was treated in a Knight-Taylor brace for 8 weeks. After physical therapy, his pain had failed to improve. The plain films demonstrate sclerosis suggestive of fracture healing *(A and B).* The alignment is acceptable, but the L1-L2 disk height is diminished. A follow-up MRI was ordered *(C).* Note the absence of significant marrow signal change or nerve root compression, but there is loss of fluid signal in the disk. Ultimately, this patient required a longer course of rehabilitation, and although he had some ongoing back pain, he was able to return to sports.
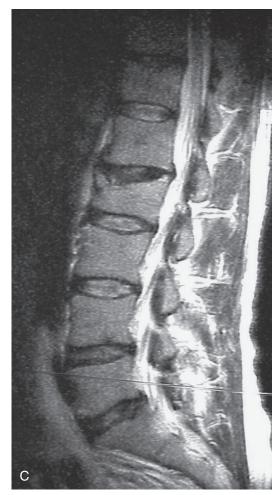
Fig. 28-8, cont'd *C*

RADIOGRAPHIC EVALUATION

Routine plain film assessment of thoracolumbar trauma includes comparison of supine and standing view radiographs. Progressive increases in Cobb angle and/or vertebral body height might predict greater instability and failure of brace management.[51,53] Concomitant splaying of the spinous processes represents additional evidence of late instability. Vacuum clefts in the bone predict poor fracture healing, and nonunion can represent Kummel's disease or vertebral body avascular necrosis.[53] Pincher or sagittal splitting injuries seem to have a higher risk of late healing problems, potentially from disk material lodged between the major bony elements of the vertebral body.[54]

The anteroposterior view must be assessed for changes in coronal balance, pedicular widening, and psoas shadow. Risser stage should be documented because younger patients are at higher risk for serious progressive deformity, such as kyphosis and scoliosis, after fracture.[55] Specialized views include cone-down views perpendicular to the fracture, which provide detailed alignment and healing information. For

patients with chronic, postfracture pain, changes in disk height should be assessed (Fig. 28-8).

Computed tomography is indicated for most moderate or high-energy thoracolumbar fractures. Carefully assess the degree of canal compromise and the status of the posterior elements. Facet distraction or overriding suggests greater instability.

In cases of thoracolumbar trauma, magnetic resonance imaging (MRI) is requested for obtunded or neurologically injured patients or to assess the ligaments. The significance of posterior edema continues to be debated. Alanay et al.[56] and others and Chow et al.[42] have separately reported that MRI findings suggestive of posterior ligament disruption might not preclude successful brace management, poor outcomes, or progressive kyphosis. Follow-up MRI, however, is useful for assessing healing. Late marrow signal abnormalities suggest nonunion.[53] Bone scans are also used to assess ongoing healing or to reveal occult skeletal injury. Scintigraphic findings evolve more slowly than do MRI marrow signals. Order a dual-energy x-ray absorptiometry scan to document bone quality in patients with low-energy fractures or radiographically apparent osteopenia (Fig. 28-9).

ALGORITHMS AND DECISION MAKING

Philosophies of spinal trauma management vary geographically and, in a given area, among physicians and institutions.[57] In general, surgical management is more aggressively pursued in Europe and the near East than in North America.[58] During the last few years, a consensus toward nonoperative management for most patients sustaining flexion-compression or axial-loading injuries of the thoracolumbar spine has evolved. Neurologic injury, ligamentous disruption, and polytrauma are the most common indications for operative intervention.[59,60]

When nonoperative management is selected, treatment protocols range from several weeks of bed rest in rotating frames to immediate mobilization without bracing or significant activity restrictions.[61,62] Although most thoracolumbar injuries do not require surgery, the literature is replete with technical details of operative management. Considerably less attention has been paid to differences in nonoperative approaches. Summary statements and review papers often lump all forms of "conservative" care into the same data block for statistical purposes.

Historically, strict immobilization was ordered on the assumption that early mobilization would risk fracture collapse and secondary neurologic deterioration. Although Hitchon and Torner[61] and Chow et al.[42] described periods of 1 to 8 weeks of bed rest after thoracolumbar burst fracture, Folman and Gepstein[63] presented a report of 85 consecutive patients who were mobilized without bracing or physical therapy. Folman and Gepstein concluded that neither bracing nor therapy was necessary (however, nearly 70% of this

cohort reported chronic pain at final follow-up). In practice, the duration of recumbency usually reflects the patient's pain rather than a parallel with fracture biology.

DECISION MAKING

Both operative and nonoperative management of spine trauma require a clear assessment of the lesion's effect on segmental stability and the ability of either surgical implants or braces to reverse that destabilizing force. To a degree, casting and bracing control motion in cases of ligamentous injury. However, the failure of reliable ligamentous healing renders bracing

ineffective as the definite management in fracture-dislocations and other translational trauma. The greater the instability is, the less appropriate bracing becomes, even as a temporizing measure. Casting and bracing are best used in the large numbers of patients who have compression injuries (compression and burst fractures) or bony flexion distraction injuries (e.g., Chance fractures) (Table 28-3).

Nonoperative management of vertebral compression fractures is almost universally accepted, considering that late neurologic sequelae are rare. The spine retains a high percentage of its preinjury stability.[28] Late kyphosis seems to be the main radiologic outcome variable. For this
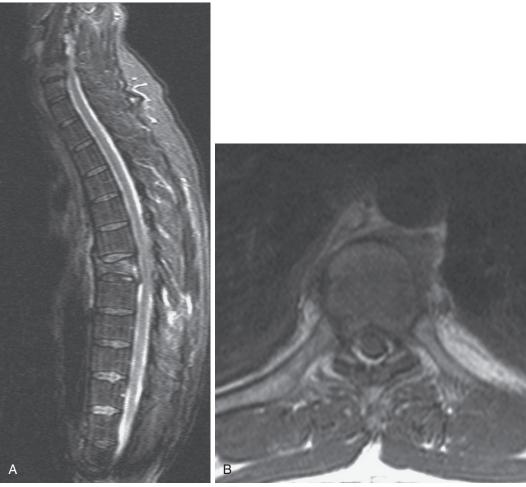
Fig. 28-9 This 42-year-old man presented reporting mid-thoracic pain after a motor vehicle accident. He was found to have thoracic radicular symptoms and an MRI was ordered, which demonstrated an acute, mid-thoracic burst fracture without marked cord compression *(A).* The axial views *(B* and *C)* demonstrate bleeding into the soft tissues anteriorly, but no marked comminution. A repeat MRI was ordered because the patient complained of ongoing pain after 4 months of TLSO bracing. On the sagittal STIR sequence, note the decreased but continued marrow signal change in the injured segment *(D* and *E).* Now, there is a signal abnormality in the adjacent level. Although these changes should alert the physician to the possibility of neoplasm, they might also reflect failure of healing and abnormal load transmission leading to endplate failure of the adjacent level. The axial image *(F)* reveals resolution of the anterior bleeding and no increase in the canal compromise with, possibly, some improvement in canal patency. The patient elected to continue nonoperative management and ultimately healed. Had operative intervention been undertaken, a sample of the vertebral body would have been sent for biopsy to look for neoplasm or osteonecrosis.

Fig. 28-9, cont'd *C, D, E,* and *F*

reason, patients with subtle compression fractures and limited pain do well with observation alone. Higher energy compression injuries involving multiple levels, greater degrees of kyphosis, or comminution at presentation are at increased risk for further collapse and are best immobilized in a full-contact cast or brace (TLSO). Limited contact orthoses, such as Jewett or CASH braces, are appropriate for intermediate injuries (Fig. 28-10).

Decision making is also affected by patient age and activity level. Post-traumatic kyphosis might not affect older, less mobile patients as markedly as it does younger highly active patients.[64] On the other hand, particularly in children younger than 13 years, some late remodeling might improve focal kyphosis after thoracolumbar compression fracture.[32]

Clinical decision making is most complex for thoracolumbar burst fractures, for which the greatest controversy and widest array of treatment options exist. The sine qua non of burst fractures is the outward comminution of fracture fragments. However, the degree to which this bursting affects vertebral height, canal patency, and overall motion segment stability is highly variable. The continuum of burst fractures includes vertebral bodies only slightly more disrupted than a compression fracture to devastating lesions with complete cord injury. This wide variability implies that a single treatment modality is not likely to apply to all burst fractures (Table 28-4).

Vertebral body comminution and fracture fragment apposition increasingly are used to assess residual vertebral body axial load-bearing capacity.[65] Although initially described by Aligizakis et al.[66] and Dai and Jin[67] as a useful predictor of failure of short-segment posterior constructs in operative stabilization of burst fractures, these criteria might be considered when predicting safe and effective brace

management. Dai and Jin recently found high interobserver and intraobserver reliability. In 2002, Aligizakis et al. used the load-sharing classification system to guide brace management in patients, arguing that the factors responsible for kyphotic collapse with short-segment, posterior thoracolumbar instrumentation were the same as those predicting failure of brace management.

The cleavage plane in flexion-distraction injuries might pass entirely through bone, entirely through disks and ligaments, or through a combination of bone and soft tissue.[68] Typically, brace management is best reserved for younger patients with slim, braceable body habitus and bone-predominant injury

TABLE 28-3 Contraindications to Bracing as the Sole Means of Stabilization in Thoracolumbar Trauma

Significant, incomplete neurologic injury*
Unstable fracture patterns†
Unbraceable body habitus
Polytrauma or injuries precluding appropriate brace selection‡
Disorders of cognition or cutaneous sensation
Poor compliance or high levels of anticipated loading§

*See text, debatable, but usually more than one root injury.
†See text, usually this list incorporates most fracture dislocations, injuries with predominantly ligamentous rather than bony disruption, distractive and many extension injuries.
‡For example, significant thoracic trauma requiring chest *PT* or a chest tube.
§In some centers, compliance problems addressed through casting or wrapping orthoses with cast material.

Fig. 28-10 This 70 year old noted thoracolumbar junction pain after a slip on black ice. He presented to his internist who ordered first a computed tomographic scan *(A),* followed by an MRI. A T12 compression fracture is noted with associated kyphosis. Note the normal marrow signal of the old compression fracture at the level below, L1 *(B).* The patient was placed in a Jewett brace after plain films revealed increased kyphosis on standing *(C).* Ultimately, the patient healed in the alignment noted at presentation without ongoing back pain.

Fig. 28-10, cont'd *B* and *C*

TABLE **28-4**   **Important Elements in Brace Selection**

Appropriate degree of immobilization (full or limited contact brace)
Appropriate segmental coverage (cranial-caudal extension)
Weight
Durability
Convenience (off the shelf vs. custom molded)
Ease of use/adjustability (particularly in patients with appendicular trauma)
Comfort/conformability
Aeration/ventilation
Pulmonary restriction (particularly in lung- or chest-injured patients)
Abdominal compression (particularly in ileus or abdominal trauma patients)

patterns. Older, more obese patients with increasing degrees of soft tissue disruption are increasingly likely to have nonoperative management fail.[69] Fracture-dislocation and ligamentous flexion-distraction injuries are not considered for brace management.[70] Along the same lines, operative intervention typically is recommended for patients with translation or rotatory instability.

If universal treatment paradigms for "standard" thoracolumbar injuries cannot be established, "correct" management of the infinite variety of hybrid and combination injury patterns remains even more contentious. The presence of more than one motion segment injury in the thoracolumbar spine has been used to argue for both operative and nonoperative care. Although some authors cite the cumulative surgical risk associated with management of multiply-injured motion segments, others claim that the risk of post-traumatic deformity also increases with multiple injuries.[13,57,71] In such cases, the treating surgeon must break the injuries down by motion segment and assess the residual stability and instability at each level.

Huang et al.[72] found that two-level injuries were rare, comprising 3.9% of the injuries treated in a consecutive series at their center. Slightly more than half were noncontiguous injuries, and the others were contiguous. The contiguous injuries clustered at the thoracolumbar junction. Most of the patients had fallen from height and were noted to have additional appendicular injury or organ injury.

TECHNICAL ASPECTS OF BRACE MANAGEMENT OF SPINAL TRAUMA

Bracing for trauma patients is most effective as part of a comprehensive management of spinal stabilization and functional restoration. The treating surgeon should effectively communicate the goals and timeline of brace wear with the patient. Cooperation, satisfaction, and overall success are optimized with frequent interactions with the patient (Table 28-5).

BRACE SELECTION AND FITTING

Orthosis selection is based on the spine's biomechanical deficit and vulnerability to deformity progression or late neurologic decline. Both over- and under-immobilization are detrimental to recovery. Braces can be custom molded or off the shelf. Custom-molded braces typically are described as more comfortable for the patient and possibly are more effective. On the other hand, they are more expensive, and several days can elapse between the casting required to create the mold and the availability of the brace itself. Off-the-shelf braces can be fitted more quickly, allowing the patient to return to ambulation more rapidly. The selection of one or the other type of brace can be predetermined by availability at your institution or the patient's insurance coverage. In other cases, the choice is based on the anticipated duration of bracing and the patient's body habitus.

Direct physician communication with the orthotist will improve bracing outcomes. The orthotist usually provides

TABLE **28-5**   **Other Portions of the Prescription for Brace Management***

1. Type of brace (or cast)
   a. Method of application
2. Duration of hospitalization
3. Duration of bracing
   a. Hours per day
   b. Weeks or months
4. Activity level and restrictions
   a. Special beds
   b. DVT prophylaxis
5. Nutritional support (including calcium and vitamin D)
6. Medications
   a. Pain management
   b. Antispasmodics
   c. Nerve agents
7. Physical and occupational therapy
   a. Modalities
   b. Extremity range of motion
   c. Gait training
8. Other modalities
   a. Injection therapy
   b. Home TENS

DVT, Deep vein thrombosis.

useful recommendations. The physician should discuss patient-specific parameters, such as polytrauma and insensate skin. If along the superior or inferior trim lines, numbness can quickly lead to deep ulceration and cessation of brace wear. Doming or relief areas can be formed into the brace to decrease pressure on wounds or surgical incisions. During warmer months, holes are drilled to improve ventilation. In some settings, direct surgeon involvement ensures that the patient is fitted with the correct brace in a timely fashion.

Next, the method of fitting is selected. Some authors recommend periods of postural reduction before fitting the brace. This reduction can occur gradually over a few days in a normal hospital bed or more acutely with the patient sedated or under general anesthesia. Common axial and flexion vectors can be counteracted with hyperextension or a lumbar bolster. A well-fitted TLSO extends cranially enough to offer significant trunk control while limiting adverse effects on the shoulder girdle. Typically, the superior trim line passes under the axilla and over the scapula posteriorly and then anteriorly to the mid-sternum. Caudal extension improves brace "hold" as well but is limited by hind-quarter mobility and the patient's need to sit without "spearing" injuries to the groin. Most often, the inferior trim line runs just superior to the ischial tuberosities posteriorly and along the inguinal fold anteriorly. A full-contact brace typically comes in two halves. Close conformation and fit are needed between anterior and posterior halves. Without a close fit, parallelogram deformation of the brace construct occurs and one side slides past the other, markedly limiting flexion and extension control.

For those braces applied without reduction maneuvers, hyperkyphosis might be decreased by donning and tightening the brace with the patient in a supine position. Although supine application is more unwieldy, elimination of the axial head and torso load on the deformity enables a more snug fit. This snug fit, wherein two fingers can just barely be inserted into the brace, is important for providing resistance to deformity. If the orthosis is donned loosely, a loss of stability and an increase in gibbus will result.

Over time, the patient is gradually weaned out of the brace. Unfortunately, little in the literature supports one mobilization plan over others.[30] Longer periods of bed rest engender cardiopulmonary decline and deep vein thrombosis (DVT) and are most appropriate for patients with markedly unstable fractures that are unable to undergo operative intervention. For such patients, bed rest in a turning frame can be required for days to weeks.[13,61]

Most typically, 1 to 2 days of relative bed rest and then brace fitting and progressive ambulation are instituted. In one study, the majority of patients were able to ambulate soon after the brace was fitted and were deemed safe for discharge after two sessions of physical therapy.[73] Typically, the physical therapy includes limitations of lifting (e.g., no-not greater than 5 pounds). We ask patients to avoid impact loading, overhead lifting, bending, and twisting. With fracture healing and return of extensor muscle function, the

restrictions are gradually lifted. A daily walking program is encouraged.

Once the patient is standing comfortably, radiographs are obtained and compared with the supine views that were obtained at presentation. Follow-up visits also include radiographic control to assess fracture alignment and healing. Often, slight fracture settling is noted with the development of mild kyphosis. This "progression" does not correlate with inferior clinical results and seldom results in any clinically detectable deformity. Although the long-term follow-up study presented by Weinstein et al.[6] concluded that "significant" late pain is uncommon and secondary neurologic deficits are rare, more significant deformity progression should prompt reconsideration of operative stabilization.

At follow-up, brace fit must be reassessed. Inspect the brace for uneven wear or cracking. The patient presenting with a brace that shows little or no wear has most likely been noncompliant. Velcro closures are a telltale area. Inspect the patient's skin.

During hospitalization, one should instruct the patient, in the clearest terms possible, how to don and doff the brace. Unfortunately, at many hospitals, only a small number of nursing and therapy support personnel assigned to assist the patient understand how to properly apply the brace. One should ask the orthotist to instruct both the patient and one shift of caregivers on the appropriate fit and donning and doffing procedure and then ask that shift of caregivers to instruct the next shift. This will provide the patient with the chance to experience the procedure several times during hospitalization and to become comfortable with the method and the appropriate degree of tightness. During that time, the patient should be instructed in means through which he or she can contact the orthotist for evolving fit and comfort issues after discharge. One should observe the patient donning and doffing the brace (or a family member assisting the patient with donning and doffing).

Significant weight changes (25−30 pounds in most adults, but less for children or smaller patients) might necessitate brace remolding. For some patients, as the fracture heals, the brace can be cut back. This will decrease effectiveness of immobilization but will ease the patient's gradual return to activity. One should remove hip spicas or convert the TLSO to a limited contact orthosis or a corset before eliminating bracing altogether.

Physical therapy is recommended for those patients having difficulty returning to activities.[73] Modalities such as muscle stimulation, heat, and massage, applied away from the spinal midline, improve muscle spasm. For patients with radiculopathy, consider skin traction, which is not a routine part of the early nonoperative care of thoracolumbar fractures. In cases of thoracolumbar trauma, skeletal traction is limited to preoperative alignment of the most highly unstable injuries.[74] Once the brace has been discontinued, a more aggressive rehabilitation program can begin. Typically, bracing continues for 12 weeks but might be used for a longer or shorter period of time depending on the patient's clinical presentation and radiographic findings. Often, over time, the midline pain over the fracture site gives way to belt-line pain along the extensor muscle insertions on the ilium. At that point, the fracture is likely stable enough to begin a core stabilization program with range-of-motion exercises.

PAIN RELIEF, MOBILIZATION, AND ACTIVITY

Even an "ideal" orthosis will not function if it is not worn. Compliance is essential for the success of nonoperative management of thoracolumbar trauma. Compliance can be improved if the patient is allowed short periods of time out of the brace. For many spine injuries, the extensor muscles can support the spine long enough to allow the patient to shower. One should explain the limited time periods so that the patient is not tempted to increase the brace-free intervals. Before returning to the brace, the patient should carefully dry off and don a clean cotton undershirt. This will improve comfort by eliminating the unpleasant sensation of plastic on skin, and, more importantly, wicking perspiration away from the body. Modern brace liners are removable and replaceable; routine cleaning and bathing can be undertaken without requiring several braces. The molded plastic of the main component of the TLSO itself is very easy to clean.

Good pain control is important in nonoperative management of trauma. Narcotic pain medications often are required during the early postinjury period. Gradually wean the medications. For patients experiencing ongoing pain symptoms, one should assess the likely source of the pain and treat it accordingly. Muscle spasm might respond to methocarbamol (Robaxin), cyclobenzaprine (Flexeril), or benzodiazepines such as diazepam (Valium). Occasionally, lidocaine patches (Lidoderm patches) or trigger point injections, particularly along the ilium, decrease muscle spasm and improve ambulatory ability.

Many patients with marked radiculopathy will consider operative intervention. In those for whom decompression is not indicated or is not possible, radicular symptoms can respond to gabapentin (Neurontin) or pregabalin (Lyrica). Alternatively, rib blocks or epidurals can be considered. Nutritional supplementation is important for all skeletal trauma patients and includes calcium, vitamin D, and vitamin C. Miacalcin nasal spray is recommended for osteoporotic patients for both its positive effects on bone metabolism and its amelioration of fracture pain. On the other hand, counsel patients to avoid nicotine and anti-inflammatories (both steroids and nonsteroidals).

Some authors have recommended casting for noncompliant patients. Others wrap a layer of fiberglass cast material around the brace to prevent patient removal. These measures should not be undertaken lightly, particularly for patients with cardiac difficulties that might require emergent access to the anterior chest wall. The medicolegal and ethical climate has shifted away from virtually all forms of patient restraint, and "compliance

enforcement" can be difficult to justify. Although Velcro and thin, lightweight, moldable thermoplastics have represented a major step forward in brace fitting and comfort, new strap and closure mechanisms will allow both greater customization from off-the-shelf braces and greater comfort from custom-molded braces. Modern LSO and TLSO designs increasingly include strapping systems designed to pull the brace inward and upward. This action improves the brace's hydrostatic effect, thereby decreasing lumbar axial loading. Similarly, these straps improve fit to limit migration and can be easily adjusted as the patient moves from sitting to standing.

Some patients with thoracolumbar trauma are first seen when they present to the emergency department. Ileus is common in patients sustaining high-energy injuries, and its possibility should be thoroughly discussed with patients who are discharged home. Often, a short period of hospitalization for observation and supportive care are appropriate. One should wait until the return of gastrointestinal (GI) function before fitting the brace or cast. Some measure of ambulation, such as bed-to-chair transfers, might be allowed. For less ambulatory patients, mechanical DVT prophylaxis and decubitus ulcer prevention and surveillance are mandatory. RotoRest and air mattress beds are helpful for less mobile patients with abnormal sensation or mental status.

Once the patient has been fitted, progressive ambulation is started with physical therapy guidance. For those with prolonged recumbency, a tilt table or gradual standing is recommended. Many trauma patients will be fairly unsteady on their feet at first, and a large brace can further impair normal balance and distort proprioception. Stair climbing should be formally assessed, as should normal transfers. Other restrictions include minimal lifting, no running or impact sports, and avoidance of prolonged sitting.

Once the patient is standing comfortably, a second, standing lateral view radiograph is obtained. For patients without significant collapse from supine to standing, activities are gradually progressed but restrictions include no bending, lifting more than 5 pounds, or twisting. The patient is discharged once voiding and eating well and having "cleared" physical therapy. The patient is seen in 2 weeks for follow-up, with instructions to present immediately if symptoms change or if leg, bowel, or bladder symptoms develop.

As the patient begins to show sclerosis at the fracture site with decreasing midline tenderness, brace wear is gradually reduced and restrictions are lifted. Once the patient is non-tender and shows restoration of range of motion and extensor muscle strength and radiographic healing, he or she is considered healed. Failure to progress in terms of pain and function can reflect bone healing issues or difficulties with rehabilitation. Unfortunately, in some settings, the patient has financial incentives to limit progress. Follow-up bone scanning and MRI can be useful to assess the degree of residual marrow signal abnormality and scintigraphic uptake, and thereby obtain a sense of the patient's stage of healing (Fig. 28-11).

OUTCOMES

One should interpret thoracolumbar trauma outcomes reports with caution. Although a number of prospective, randomized clinical series have recently been reported, the indications for operative and nonoperative management have varied widely among them. Other variables include periods of bed rest before bracing, duration of bracing, types of braces used, and patient activity restrictions while in the brace. When counseling a patient regarding likely outcomes, one should emphasize the unknowns, which include the significant variability in patient compliance with bracing instructions and postoperative rehabilitation. Older outcomes reports favor radiographic rather than clinical measures.

OUTCOMES OF BRACING VERSUS FRACTURE TYPE

Most of the available reports detailing outcomes of brace management of thoracolumbar fractures describe compression or burst injuries. Although most fracture dislocations should be managed operatively, an intermediate category of flexion-distraction injuries, the Chance or "seat belt" fracture, can heal after brace immobilization. In 1948, Chance[75] reported good results in the treatment of lap belt fractures through osseous tissue with hyperextension casting. Forty years later, the recommendations remain unchanged.[76] Thus, controversies relate to ongoing disagreements regarding what "stable" means for burst fractures.

FRACTURE ALIGNMENT AND KYPHOSIS

Fracture alignment and kyphosis issues impact decisions around bracing for thoracolumbar injuries in two related ways. First, the debate over the relationship between fracture deformity, final sagittal spinal balance, and late pain and dysfunction continues.[6,77] Second, if some amount of collapse or increased kyphosis is responsible for late pain, nonoperative management should not be considered for patients presenting with deformity exceeding that level.

In a classic paper from 1988, Weinstein et al.[6] presented a report of 42 burst fractures treated nonoperatively with a body cast, brace, or bed rest. In most cases, the authors found that brace management of burst fractures without neurologic deficit had acceptable long-term results. Included in this series was a number of patients with relatively severe focal kyphosis. Outcomes were not found to be significantly related to the final sagittal contour or degree of focal kyphosis. On the other hand, Gertzbein[77] reported the results of a multicenter trial organized by the Scoliosis Research Trial in which 1019 patients with spinal fractures under the care of 64 physicians from 12 countries were followed prospectively for 2 years. Based on the data, Gertzbein concluded that a kyphotic deformity of greater than 30 degrees at 2-year follow-up was associated with an increased incidence of significant back pain. Overall, patients who had surgery

complained less of severe pain than did those who were treated without surgery.

More recently, the amount of vertebral body collapse has been thought to be less important than spinal alignment. For example, in a series presented by Folman and Gepstein,[63] 85 patients were treated with a short period of bed rest and pain intensity was related to the angle of local kyphosis but not to the percentage collapse of the vertebral body. The impact of post-traumatic kyphosis on long-term back pain, however, remains poorly understood and continually debated. Several recent series have failed to find a correlation between kyphosis and clinical outcome.[30,42,56] Others reported that poor final sagittal balance adversely affected clinical outcomes.[78,79] Vaccaro and Silber[80] reported that the single greatest impediment to functional recovery in patients who are under-immobilized for thoracolumbar fractures is post-traumatic kyphosis. Similarly, the numerous reports outlining benefits of corrective surgeries for chronic post-traumatic hyperkyphosis suggest that excessive kyphosis is not trivial.[81−83]

Considering our limited understanding, if kyphosis leads to functional problems or pain, the question of why this deformity ostensibly causes pain in only a subset of patients is less clear. Often, muscular factors are blamed for ongoing pain and fatigability. Neurologic problems can include spinal cord draping over the apex of kyphosis.[80] Disk level changes suggestive of post-traumatic degeneration have also been blamed. These changes were studied by Oner et al.[84] in a comparison study between 26 nonoperatively and 37 fixator-interne treated patients with thoracolumbar burst injuries. Most disks showed predominantly morphologic changes with no variation in signal intensity. In operatively managed

Continued

Fig. 28-11 This 63-year-old patient presented to the emergency room after a fall from a ladder complaining of back pain and numbness, but no pain in an L3 radicular distribution to the anterior thigh. There was no weakness or leg pain. Radiographs *(A* and *B)* demonstrated an L3 burst fracture in the context of lumbar spondylosis and an L3-L4 spondylolisthesis. After 2 days, the patient was fitted for a custom molded TLSO *(C* and *D)*. At 6-week follow-up, the patient is noted to have hypolordosis but no significant collapse of the fracture itself *(E)*. The radicular symptoms had resolved and the pain was improving rapidly. By 12 weeks *(F* and *G)*, the patient's fracture appeared to have incorporated. He was no longer tender over the bone. The brace was discontinued and physical therapy was instituted.

Fig. 28-11, cont'd *C, D, E, F* and *G*

patients, recurrent kyphosis seemed to result from disk creep into the central depression of the bony endplate rather than from degeneration per se.

How the treating physician interprets the conflicting literature on deformity will define his or her approach to individual patients. For example, for physicians unconvinced by the data suggesting that kyphosis leads to pain, bracing or casting is undertaken merely to limit motion at the injury level to speed healing rates and decrease pain. Other surgeons take a "less is more" approach and seek to restore, as closely as possible, an anatomic alignment. For these doctors, bracing often is combined with reduction maneuvers to improve alignment. Strict brace wear that maintains reduction to further limit kyphotic collapse during fracture healing is prescribed. Under this philosophy, the question becomes, how well do braces work to control kyphosis?

In 2000, Ohana et al.[62] undertook a retrospective, matched cohort comparison of braced and not-braced patients undergoing nonoperative management for axial loading injuries and less than 30% height loss. They found no difference in collapse or outcomes. Most studies show significant early reduction only to report gradual collapse to the index deformity by final follow-up. For example, in a 2003 series presented by Tropiano et al.,[41] for 45 patients treated by closed reduction and casting for thoracolumbar burst fractures, closed reduction resulted in significant correction of vertebral wedging, from a mean of 15 to 5 degrees. However, the deformity tended to recur by 4 months. Similarly, at the 34.3-month follow-up of 26 nonoperatively-treated patients, Chow et al. reported that kyphotic deformity was corrected with hyperextension casting but tended to recur during mobilization and healing.

In a series of 15 patients prospectively assessed for a mean of 31 months for sagittal balance after nonoperative management of thoracolumbar fracture, each patient was sedated and placed in a total body cast.[56] The casts were removed at the end of 3 months, and no further external stabilization was applied. The average local kyphosis angle after trauma 16.5 degrees (range, 0−34 degrees) was corrected to 5 degrees (range, 19−25 degrees). By the third month, the angle had deteriorated to 14.6 degrees (range, 2−25 degrees) and to 17 degrees (range, 2−29 degrees) at final follow-up. A similar tendency existed for both sagittal index and percent anterior body height.

By this rationale, patients with marked kyphosis should be offered operative stabilization. Yet recent randomized trials comparing posterior stabilization and brace management can be criticized for the limited treatment effect in their operative arms. Standard, short-segment transpedicular constructs show similar recurrence of deformity to that recorded after bracing.[1,85,86] In a comparison study by Andreychik et al.,[78] between 25 operatively and 30 nonoperatively-managed patients, at a mean follow-up duration of 79 months, no difference in kyphosis was observed between braced patients and those undergoing short-segment transpedicular instrumentation.

The limited kyphotic control afforded by posterior stabilization led the authors to recommend anterior reconstruction for patients with marked kyphosis or comminution. Considering the profound differences in morbidity between bracing and anterior corpectomy for a burst fracture in a neurologically intact patient, more convincing evidence of the association between kyphosis and long-term dysfunction would be helpful. Yet for most surgeons in North America today, excessive kyphosis at presentation represents an indication for operative intervention.

Beyond those patients presenting with significant deformity are those who experience collapse during the follow-up interval. It remains difficult to predict which fracture patterns are likely to collapse. Recent studies have reported significantly different collapse patterns among mid-thoracic, thoracolumbar, and mid-lumbar fractures.[79,87] Although some patients report severe ongoing pain, many more have radiographic evidence of collapse in the context of improved pain and function. Convincing these patients to undergo operative stabilization 8 weeks after injury might be difficult. At the same time, the concept of bracing it and operating later if collapse occurs is not justified as a means of fracture management because of the challenging nature of late surgery in this patient population.

At that point, a reasonable middle course includes close radiographic follow-up and detailed discussions with the patient and family about both operative and nonoperative approaches. Younger, more active patients might benefit from more aggressive attempts to maintain or regain sagittal balance.

NEUROLOGIC RECOVERY AND DECLINE

Most fractures associated with major neurologic injuries are operatively treated. Some surgeons continue to worry that brace management might allow secondary fracture collapse with increased retropulsion and neurologic decline. This concern has produced recommendations for surgery based on the degree of retropulsion or stenosis. Yet, neurologic decline in braced thoracolumbar fractures is rare. Of three recent well-conducted studies that included large numbers of patients treated in braces for major thoracolumbar trauma, not one case of neurologic decline occurred.[1,41,42]

On the other hand, in 1994, Gertzbein[88] reported neurologic decline in 35 of 1019 patients during hospitalization. Neurologic instability was associated with a rotational component to the burst fracture. It is possible that the rotational injuries were mechanically most like fracture-dislocations and required operative stabilization.

In animal studies, decompression of the spinal canal improved neurologic outcomes.[89] Operative intervention for "canal clearance" remains popular in an effort to improve to neurologic outcomes and prevent late decline. However, the recent literature ascribes the critical neurologic injury to the moment of impact. Subsequent changes in neurologic status are related to secondary injury cascades or healing. The

fracture fragments' position on subsequent imaging has little impact on recovery.[90] Currently, data confirming the benefits of early decompression observed in controlled environments of simulated trauma in animal models has not been forthcoming in human studies.[90] Yet, many practitioners use the animal data to justify early (and sometimes emergent) canal decompression in spinal cord or cauda equina injuries.[91,92]

Even for proponents, however, the degree of canal occlusion is often not related to the severity of the neurologic injury.[93] Gertzbein[88] report that, although nonoperative treatment was associated with improved neural function, anterior decompression led to more rapid and complete recovery. Critics cite methodologic flaws in this and similar studies and report equal neural recovery with nonoperative management.[94] Reports that late decompression was associated with further improvement in neural function has been used by both proponents and opponents of early canal decompression.[92,95] In 2000, Boerger and colleagues[90] reviewed 275 burst fracture management publications. Of these, 60 met minimal inclusion criteria and were analyzed. Pooling outcomes data failed to establish neurologic benefits from surgical decompression. On the contrary, significant complications, including neurologic decline during or after surgery, were reported in 75% of papers, including neurologic deterioration. The authors concluded surgery should be performed for stability only.

Similarly, in 2001, Dai retrospectively analyzed 31 patients with thoracolumbar burst fractures. In this group, 7 were left untreated while 16 had been treated nonoperatively and 8 underwent operative reduction and stabilization. During the 3- to 7-year follow-up intervals, initial and final degrees of neurologic deficit were compared to the stenotic ratio of the canal. The degree of canal stenosis significantly decreased from first examination (range, 12.3%–74.5%; average, 26.2%) to final follow-up (range, 5.4%–46.5%; average, 19.2%). Although operative decompression improved the stenosis ratio earlier, this difference had little bearing on ultimate neurologic recovery, which was highly significantly related to stenotic ratio at first examination only.[96]

PAIN AND FUNCTION OUTCOMES

Patient-centered outcomes literature of nonoperative spine fracture management is increasing but overall remains sparse. Similarly, the first direct comparisons of operative and nonoperative treatment have only recently been published. Classic papers from the 1980s include McEvoy and Bradford's[5] report on nonoperative treatment of burst fractures. The authors described a treatment regimen including a mean of 4.5 months of polypropylene bracing or casting (range, 2–6 months) and favorable objective and radiologic outcomes. Willen et al.[97] also reported good radiographic outcomes with hyperextension bracing as part of a nonoperative treatment regimen for fractures in the thoracolumbar region. More recent series have included patient-centered, subjective

measures, and have reported generally favorable but highly variable results of bracing for thoracolumbar trauma. In both Chen's series, one of 26 patients was dissatisfied. In Alanay's[56] series of 15 patients followed for 31 months, all were satisfied with management consisting of 3 months of body cast immobilization.

Despite overall good satisfaction scores, residual pain levels can vary from none to "constant, severe pain" in radiographically similar injury patterns. In 2003, Tropiano and coworkers[41] reported the retrospective outcomes of 41 neurologically intact patients with thoracolumbar and lumbar burst fractures treated by closed reduction and casting. At 8 months, 64% had minimal or no pain whereas 8% reported constant, severe pain. Chow and others[42] found, at a mean 34.3-months follow-up of 26 patients treated nonoperatively, 79% reported little or no pain. Few objective characteristics distinguished patients with and without ongoing pain issues.

On the other hand, in McLain's[98] series, 5-year outcomes in *operatively* managed fractures, persistent back pain generally stemmed from an identifiable and correctable mechanical problem such as sagittal imbalance, pseudarthrosis, or persistent instability. Ostensibly, ongoing pain issues in the nonoperatively treated population could stem from similar mechanical issues, particularly sagittal balance.

One more objective form of outcomes assessment in trauma patients is their rate of return to work. Proponents of nonoperative care cite iatrogenic factors associated with operative treatment that might impede recovery, including: dissection of paraspinous muscles, damage to motion segments (i.e., fusion), and the implantation of spinal devices. Unfortunately, return to work parameters in nonoperatively-managed patients are similarly variable. In Tropiano et al.'s [41] series, 71% of 45 patients were employed at the time of injury. At 8-month follow-up, 58% of those patients had returned to employment. Chow et al.[42] found that 75% of his patients had returned to work without restrictions after nonoperative management. In an outcomes review in a cohort of U.S. Army aviators, 77% recovered sufficiently to return to aviation.[99]

In Folman's series of 85 patients treated with a short period of bed rest (i.e., without bracing or surgery), 25% had changed jobs, typically from full time to part time. They found that 48% of those that had filed lawsuits related to their injuries were out of work at the 6-month mark as opposed to 11% who had not filed suits. In 1996, Burnham and others[100] analyzed their prospective cohort of spine fracture patients for factors predictive of employment 1 year after injury. At 1 year, 54% of their patients were working. A higher percentage of patients were working in a limited or part-time capacity than preinjury. Employment at the time of injury and an absence of worker's compensation coverage were found to be predictors for continued employment.

In comparison, McLain[98] reported 5-year outcomes in a series of 70 patients undergoing operative management for

burst fractures, more than half of which were associated with major neurologic injury. In this group, despite the severity of the spinal and concomitant injuries, 70% returned to full-time work. Of those working, 54% returned to their previous level of employment without restrictions; 16% maintained full-time, but lighter duty, jobs. Twenty-two percent were working part-time or not at all and 8% were unemployed despite unrestricted functional status. In his series, McLain found that work status correlated directly with neurologic impairment ($p < .00005$) and was not related to level of injury, hardware failure, extent of surgical dissection, or construct pattern.

DRAWBACKS AND COMPLICATIONS OF BRACING

One of the keys to successful brace management lies in close patient follow-up. It might seem counterintuitive, but brace treatment typically requires more intensive management than operative intervention (Table 28-6).

SKIN AND SUBCUTANEOUS TISSUE INJURY

Although data enumerating rates of ulceration under braces remains limited, skin problems are likely the most common complication of bracing and the most frequent cause of early brace cessation or noncompliance.[101]

All braces require some cutaneous pressure through which they apply force to the spine. Excessive pressure leads to ischemia with pain, breakdown, or ulceration.[41] Pressure, of course, refers to force per unit area. Therefore, pressure levels can be minimized by limiting the force applied or spreading it over a wider area. The final common pathway to perfusion disruption and secondary ischemia is microvessel occlusion, which requires only 32 mmHg of force.

Some patients face much greater skin breakdown risk than others. At-risk patients include those with limited mobility or prolonged recumbency, altered sensation or sensorium, skin shear or friction, local moisture, and those with poor nutrition or hydration. As with the pressure sores seen in paraplegic patients, even regional areas of diminished sensation disrupt the normal exteroceptive feedback loop. The normal discomfort from ischemia or pressure no longer stimulates subconscious shifting of position to unload the dependent part. Patients with sensation abnormalities and their families should be taught how to examine the skin and what stage 1 pressure sores look like. Home nursing visits and close-office follow-up should be arranged. In patients with poor nutrition, protein wasting leads to tissue edema, altered tissue repair, and regional compromise of the antimicrobial mechanisms. Edematous tissue, moreover, has impaired oxygenation and decreases elasticity.[102]

Prolonged exposure of the skin surface to moisture compromises its barrier functions. For example, wound drainage,

TABLE 28-6   Complications of Bracing
Failure of stabilization
Incorrect orthosis for level of injury
Incorrect orthosis for mechanism or vector of injury
Incorrect orthosis because of patient-specific factors (sensation loss, associated injuries, body habitus)
Unsuspected spinal instability (e.g., fracture dislocation misidentified as a compression fracture)
Skin and soft tissue problems
Pain
Dermatologic problems: contact dermatitis, ingrown hair
Skin-wound breakdown
Superficial nerve compression (lateral femoral cutaneous, cluteal)
Muscle atrophy
Contracture formation
Associated joints: shoulders and hips
Skeletal problems
Brace-associated osteopenia
Increased motion at end-segments above and below brace
Thoracic and abdominal problems
Ileus
Cast syndrome (superior mesenteric artery syndrome)
Restrictive pulmonary dysfunction
Vascular problems
Varicose veins
Extremity edema
Hemorrhoids
Psychological dysfunction
Brace reliance
Secondary gain
Adjustment disorders

incontinence, and sweating overhydrate the skin's outer layer, reducing its mechanical strength and resistance to bacterial permeation. Later, tissue pH changes lead to sloughing and deep layer exposure, which increases underlying dermal susceptibility to infection and ulceration.

Both shear and moisture below the brace can be limited by using a cotton undergarment between the skin and the brace. This garment should be changed at least daily and more often in hot weather or tropical climates. Hygiene has a significant role and access to the underlying tissues is a major advantage of removal bracing as opposed to spinal casting. For higher risk patients, when possible, use a limited contact brace.

Bony prominences with poor fat and muscle coverage, such as the sacrum, greater trochanter, and thoracic spinous processes are particularly vulnerable to skin breakdown. To accommodate them, especially in very thin patients, these areas must be padded or a hole cut in the brace. Brace wear

can also affect subcutaneous tissue. Muscle and fat are actually more vulnerable to focal ischemia than skin. These tissues might develop necrosis earlier and more extensively. This makes the risk to bony prominences seem paradoxical. In truth, fat and muscle are more important for dissipating force than skin or bone. Problems usually occur at abrupt transition points between the muscle and bone, which maximizes force at that point.

Pressure atrophy of subcutaneous fat can lead to redistribution of deposits. In one series, trochanteric fat pads were found in 23% of 300 young female subjects using Boston brace.[103] These changes lead to significant cosmetic deformity and also limits the continued effectiveness of the brace.

MUSCULOSKELETAL AND CARDIOPULMONARY CHANGES

Aside from pressure atrophy, lack of normal motion and tone leads to disuse muscle atrophy rapidly after even brief periods of bracing.[102] The associated weakness and loss of muscle bulk can be profound. In some cases, the patient will have to be gradually weaned from the brace with an active rehabilitation program.

Particularly in neurologically-injured or sensorium impaired patients, contracture must be avoided. Contracture can occur from simple joint immobilization or from imbalanced effector forces across the joint. Over time, the affected muscles become painful and permanently fibrotic. These changes are more commonly noted in the axial skeleton after halo management but also seen, albeit to a lesser degree, with long periods of TLSO management. Long braces also affect shoulder and hip girdle function.[47]

Similarly, the mechanical impediments related to bracewear affect cardiopulmonary function. Although this effect is more pronounced with halo braces, it is seen to a more limited extent with TLSO wear. In both, the brace is a mechanical impediment to venous return and normal ventilatory mechanics. For younger, healthy patients, this might not be an issue. On the other hand, patients with underlying cardiac or pulmonary disease are at higher risk, the extent of which has not been quantified. Incentive spirometry can be recommended for all patients. Pulmonary function tests should be considered in higher risk patients.

TLSO increases risk for lower-extremity venous stasis with varicose veins, hemorrhoids, and most dangerously, DVT. Activity, including a walking program, should be encouraged. Initially, a compression hose might be appropriate, especially in patients in a thigh cuff. Later, in certain at-risk patients, anticoagulation can be considered. Pulmonary complications increase in more cranial injuries, particularly in patients with spinal cord injury (SCI).[104] Particularly in patients with major neurologic injury and higher thoracic injury, the risks of bracing might outweigh its benefits.

GI DYSFUNCTION

Abdominal compression, especially when combined with spinal hyperextension, stretches the superior mesenteric artery across the ventral duodenum. This compression can lead to a postprandial bilious emesis, epigastric pain, and distention and has been termed cast syndrome.[105] Recent weight loss, recumbency, posturing, and spasticity all increase the risk of cast syndrome development.

In patients with GI irregularities associated with brace wear, testing includes a barium swallow, which shows duodenal compression with delayed passage into the small intestine. Treatment begins by removing the brace. Some patients might need endoscopy for gastric decompression, parenteral alimentation, or enteral feeding with a tube passed beyond the duodenum. Occasional, refractory cases require surgical release.

More typically, bracing can delay diagnosis in patients with significant intra-abdominal injury. Particularly in casted patients, serial abdominal examination is difficult. Risk factors for abdominal injury include Chance fracture pattern, abdominal wall contusion, and lap belt use. In these patients, the risk of serious intra-abdominal injury is at least 22%.[106] Prior to bracing, serial physical examinations should be conducted until normal GI function returns. Consider computed tomography of the abdomen and diagnostic peritoneal lavage for patients who are being considered for casting and those for whom follow-up can be problematic.

PSYCHOSOCIAL DYSFUNCTION

In up to 3% of braced patients, psychological problems are noted. Despite the selection bias of a trauma patient pool, this rate is not significantly different from that seen in adolescent girls undergoing brace management for scoliosis. In the latter group, body issue images predominate. In the trauma group, claustrophobia, anxiety, and adoption of the sick role are seen. In patients with active worker's compensation or litigation issues, the brace might be seen to connote legitimacy of the medical claim.[22]

Psychosocial dysfunction has a number of implications on patient outcomes but can affect treatment more directly in the form of suboptimal compliance.[107] Patients should be weaned from their braces as soon as fracture and soft tissue healing allow.

COMPLICATIONS DESCRIBED IN THE RECENT BRACING LITERATURE

Although descriptions of potential complications related to brace management abound, aside from skin problems, most other complications are rare. Most series of nonoperative care emphasize radiologic findings such as kyphosis and functional and pain outcomes with little discussion of the complications of brace management. Many papers will simply

state that no significant complications were associated with their treatment protocol.[66] When data are available, it is important to understand the protocol used because approaches ranging from immediate ambulation with no brace and 6 weeks of recumbency in a kinetic bed all fall under the rubric of "nonoperative" care.

In Tropiano and coworker's[41] series of 45 patients with thoracolumbar and lumbar burst fractures treated by closed reduction and casting, no complications were noted from closed-fracture reduction maneuvers or casting. The authors concluded that closed reduction and casting of thoracolumbar and lumbar burst fractures is a safe treatment method that yields acceptable functional and radiographic results.

Shen and coworkers[108] prospectively compared outcomes and complications in 47 patients treated in a hyperextension brace with early ambulation versus 33 patients who underwent three-level spine fusion. Although the operatively-treated patients had earlier pain relief, the outcomes at 2 years were the same. None of the nonoperative patients worsened neurologically. Final kyphosis angle was similar as well, because operatively achieved reductions were found to be lost during the follow-up period. In the nonoperatively-treated group, kyphosis worsened a mean of 4 degrees, but retropulsion decreased from 34% to 15%. Hospital charges were four times higher in the operative group. There were no complications in the braced group. Interestingly, the same authors examined their experience with nonoperative management in a group of 38 patients with single-level burst fractures that included injury of the posterior column. In these patients, no brace was used and immediate activity was allowed as tolerated. The mean increase in kyphosis was 4 degrees and the maximum increase was 6 degrees. In this group, complications were limited to transient urinary retention.[45]

In 1999, Rechtine and others[109] compared the complications of 235 patients with thoracolumbar spine trauma of whom 117 underwent operative stabilization and 118 underwent "aggressive nonoperative" treatment including 6 weeks on a kinetic bed. The authors found no difference in the rate of decubitus ulcers, DVT/pulmonary embolism (PE), or mortality. Deep wound infections were found in 8% of the operative cases.

In Wood et al.'s [1] prospective, randomized comparison of operative versus brace management for thoracolumbar burst fractures in 47 consecutive patients, the rate and types of complications between the operative and nonoperative groups were "distinctly different." Sixteen of the 24 surgically treated patients were found to have 19 complications. On the other hand, 2 complications were seen in 3 of the 23 patients treated nonoperatively. The complications seen in the nonoperatively treated group were, in general, less severe than those seen in the operatively-treated group. In this series, one-patient with Parkinson's disease was not able to tolerate the brace.

CONCLUSION AND FUTURE DIRECTIONS

Future directions for bracing in thoracolumbar fractures include improvements in brace design and comfort. Improved materials design and manufacturing will allow multilayer designs in which stiff layers alternate with more compliant layers internal to the brace and more compliant external layers that improve fit and comfort. These alternating layers can allow a modularity that can adjust stiffness to activity level (e.g., sitting up and reading versus physical therapy versus in the car). This modularity can be extended via step-down options.

Given modern medicolegal concerns, many physicians have expressed more than a research interest in monitoring patient compliance with their bracing prescriptions. A number of intrinsic pressure monitors with data loggers have been demonstrated effective as compliance monitors, but the clinical impact is not yet clear.[107] At this point, such compliance monitors are not widely available, but brace use can be assessed through careful inspection of the brace. Finally, future trends might include hybrid procedures in which percutaneous injection of calcium phosphate or other resorbable bone cements is combined with bracing to provide comfort, protection, and stability.

Today, thoracolumbar fracture care appears to be in midst of a swing away from operative management and toward more conservative regimens including bracing, bed rest, and activity modification.[110] In the 1980s, advances in spinal imaging allowed treating surgeons to visualize disrupted anatomy in new ways and this lead to increased rates of surgery to "fix" the problem.[58] More recently, randomized and controlled studies have clearly demonstrated that, despite "middle column" disruption for example, surgery does little to "protect" the patient from secondary neurologic decline or even significant kyphosis. In fact most stable thoracolumbar injuries are amenable to nonoperative management.[1,108,111]

On the other hand, we lack consensus over what constitutes a "stable" or "unstable" thoracolumbar fracture. Patients with major neurologic or ligamentous injury tend to be treated operatively. Neurologically intact burst and compression fracture patients, on the other hand, are best approached with a plan for nonoperative care. The decision to operate can then be based on coexisting injuries or failure of brace management, rather than a de facto operative strategy.

Hopefully, the next few years will yield clarification as to the role post-traumatic kyphosis plays in long-term back pain and function. Current data cannot justify a firm radiographic cut-off for either canal stenosis or kyphosis beyond which operative intervention is "needed." That operative management should further emphasize the need to justify these procedures with outcomes data.[112] On the other hand, the "personality" of the fracture including the level of energy imparted, anterior column comminution, associated trauma, and patient braceability might give

some clues as to the likelihood of success with nonoperative management.

In either case, bracing represents an *active* treatment modality in which close patient-physician cooperation is needed in selecting the appropriate cast or orthosis, outlining hours of use and activity modification, and close follow-up physical examination and radiographic control.

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Surgical Approaches for the Operative Management of Thoracolumbar Spine Fractures

The surgical approach chosen for the treatment of thoracolumbar trauma depends not only on the location of the fracture but also on the nature of the injury itself. The more familiar posterior approach most often is used for deficits of the posterior column, such as flexion-distraction type injuries and fracture dislocations, whereas the anterior approach typically is reserved for defects in the anterior column or space-occupying lesions involved with neurologic deficits, such as burst fractures. On occasion, a variant of a posterior approach—posterior-lateral, transpedicular, and costotransversectomy—can effect decompression of the anterior column via a posterior approach.

RADIOLOGIC IMAGING

Plain radiographs provide insight into the general pathologic condition and the general alignment for preoperative planning. Modern emergency department and trauma resuscitation systems use high-speed computed tomography (CT) that quickly allows visualization of the spinal axis with reconstruction in the coronal, sagittal, and axial planes. CT is considered the examination of choice with which to best visualize the fractured bony elements.

Magnetic resonance imaging (MRI) is being used with increasing frequency to assess the soft tissues of the injured spine, such as the intervertebral disks and posterior osteoligamentous complex and also the spinal cord and nerves themselves, and is really the treatment of choice in this area. Myelography with post-myelogram CT is an alternative tool that is especially useful in instances of indwelling metal or instrumentation or in cases of severe spinal deformity.

SURGICAL APPROACHES

The surgical approach for thoracolumbar trauma is dictated principally by the location of the pathologic abnormality. Traumatic pathologic abnormalities of the posterior elements most commonly are approached from a posterior incision, whereas vertebral body deficits more commonly are treated from an anterior approach.[1–4]

SURGICAL APPROACHES IN OUTLINE FORM

Upper thoracic spine
    Posterior
    Transthoracic
        Transpleural or retropleural
    Anterior sternotomy
    Thoracoscopy
Mid thoracic spine
    Posterior
    Transthoracic
    Costotransversectomy
    Thoracoscopy
Thoracoabdominal
    Posterior
    Posterior-lateral
    Transpleural retroperitoneal
    Transpleural transdiaphragmatic
    Retropleural retroperitoneal
    Transpedicular (egg shell)

POSTERIOR APPROACHES

The primary indications for a posterior approach to the thoracic or thoracolumbar spine are for laminectomy and fusion with or without instrumentation. The fracture categories most commonly treated with this approach are simple or multiple compression fractures, stable burst fractures, flexion-distraction injuries to the posterior osteoligamentous complex (seatbelt or Chance-type injuries), or fracture dislocations with either no neurologic injury or complete neurologic deficit.

These incisions are straightforward and familiar to the spine surgeon. Posterior approaches tend to take less time,

involve less blood loss, and be less expensive.[5] This approach affords exposure of the spinous processes, the laminae, the facets, the pars interarticularis, and the transverse processes (Fig. 29-1). Although the anterior aspect of the spinal canal is difficult to visualize with a laminectomy, a transpedicular approach can provide circumferential visualization of the canal for decompression, if necessary, although the midline anterior dura remains inaccessible to direct visualization.

The patient typically is placed prone on a frame with the abdomen and thorax free from pressure. Bony prominences, especially the anterior-superior iliac crest, should be well padded to prevent postoperative paraesthesias. Intraoperative monitoring can be used but is not necessarily mandatory. Intraoperative C-arm might aid in pedicle screw placement.

The incision is extended at least two levels above and two levels below the fractured level. Electrocautery aids in extending the dissection laterally to the lateral extent of the spinous processes. A three- or four-point fixation bending moment with cantilever forces to restore the normal sagittal alignment is used. Indirect reduction of any retropulsed bony fragments can be accomplished by using a gentle combination of distraction and lordosis as long as the fracture is a recent one. Once accomplished, fusion and instrumentation with hooks, wires, or pedicle screws can be easily achieved.

TRANSPEDICULAR APPROACH

Although the transpedicular approach was first described as a means of removing extruded thoracic disk herniations, when performed in a bilateral approach, complete diskectomies and vertebrectomies can be performed,[1,6–8] as can posterior instrumentation, all through a single approach (Fig. 29-2).

The transpedicular approach (eggshell procedure) is a somewhat specialized treatment of anterior pathologic abnormality via a single posterior approach.[9] It might be useful to affect anterior decompression of both sides of the spinal canal in the setting of a kyphotic deformity, especially if surgery has been delayed for any reason. It is useful for treating older patients and those who might not tolerate an anterior transthoracic or transdiaphragmatic approach.

The posterior spine is exposed in the usual manner, the spinous process and laminae are removed or thinned, and the cortical bone over the pedicles is removed. Using successively larger curettes, the pedicle is enlarged, allowing access into the vertebral body (Fig. 29-3). Angled curettes are then used

Fig. 29-1 Posterior approach affords exposure of the posterior elements out the lateral aspect of the transverse processes. (Reproduced with permission from Kim DH, Jahng TA, Balabhadra RS, et al: Thoracoscopic transdiaphragmatic approach to thoracolumbar junction fractures. Spine J 4:317–328, 2004.)
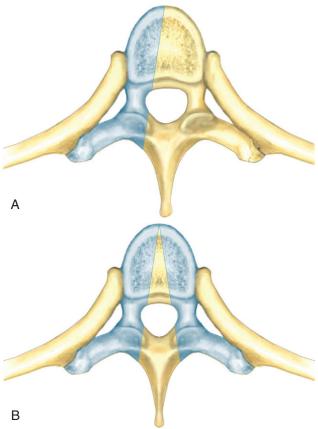

A

B

Fig. 29-2 Schematics illustrate the amount of bone that can be removed via a unilateral transpedicular approach (A) and from a bilateral approach (B). (Reproduced with permission from Khoo LT, Beisse R, Potulski M: Thoracoscopic-assisted treatment of thoracic and lumbar fractures: A series of 371 consecutive cases. Neurosurgery 51 [suppl 5]:S104–S117, 2002.)
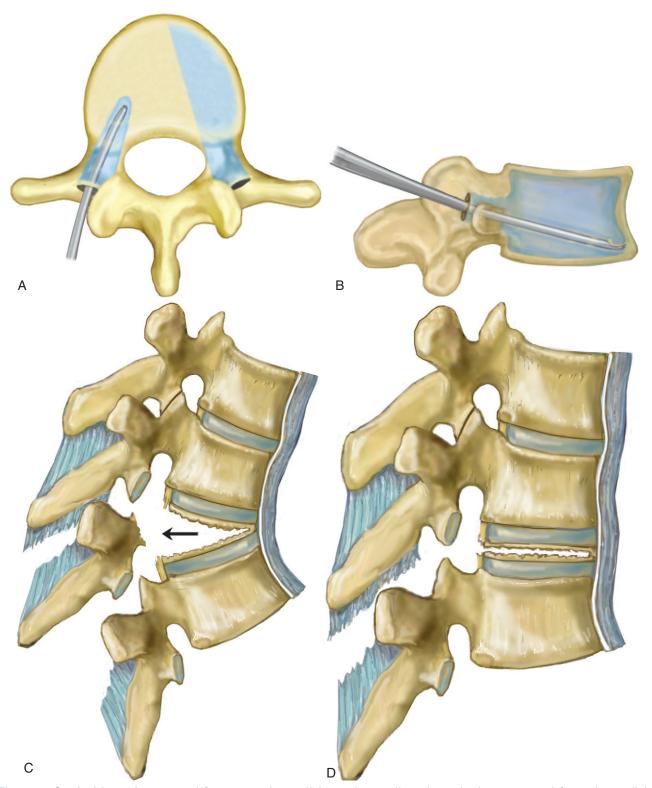
A

B

C

D

Fig. 29-3 Cortical bone is removed from over the pedicle, and cancellous bone is then removed from the pedicle extending into the vertebral body *(A)*. Curettes can next be used to remove cancellous bone from within the vertebral body, leaving the cortical (egg shell) *(B)*. Once the cancellous bone is removed, the posterior wall can be either removed or pushed into the vertebral body *(C)* and the deformity can then be corrected *(D)*. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437–1470.)

to remove cancellous bone from within the vertebral body to the limits of the vertebral walls. The pedicle on the contralateral side is similarly approached. The contents of the spinal canal are protected by the posterior vertebral body wall and the lamina. If retropulsed bone is present, it might be forced back anteriorly into the cavity of the vertebral body created by the curettage. After that, instrumentation can be applied and the deformity corrected through a controlled collapse of the osteopenic fractured body (Fig. 29-4).

POSTEROLATERAL APPROACHES

COSTOTRANSVERSECTOMY

The costotransversectomy approach provides access to the posterolateral aspect of the vertebra, if necessary, and to the posterior elements.[3] It is especially useful in the upper thoracic spine, where a transthoracic approach to the anterior spine is more difficult. The approach can be either transpleural or extrapleural.

To affect the costotransversectomy, the patient can be either prone or in a lateral decubitus position. In the upper thoracic spine, the incision is vertical midway between the spinous processes and the scapula (see Fig. 29-4). In the lower thoracic spine, the distal aspect of the incision can be extended obliquely, following the rib to be approached. Once the paravertebral muscles are retracted and the costotransverse joints and transverse processes exposed, the rib can be cut at a distance of approximately 6 to 8 cm from the costotransverse joint. The costotransverse joint ligaments are released and the pleura protected before cutting the rib and removing it (Fig. 29-5). If the transverse process is removed,
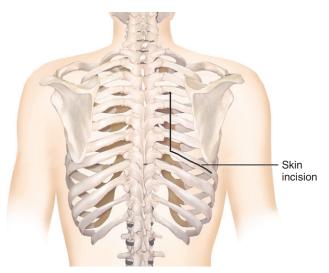

Fig. 29-4 Approach for costotransversectomy. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437–1470.)
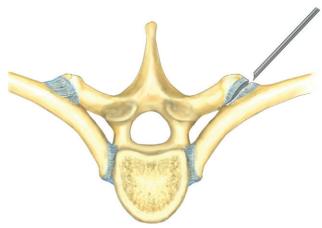
Skin incision

Fig. 29-5 Diagram detailing the release of the rib from the undersurface of the transverse process during a costotransversectomy. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437–1470.)

access to the neural foramen is facilitated. From that point on, the lung and pleura are protected by malleable retractors. Typically, a one- or two-rib costotransversectomy allows visualization of up to three vertebrae.

ANTERIOR APPROACHES

The advantage of an anterior approach to the injured spine is the ability to not only reconstruct the anterior weight-bearing column, especially in the setting of severe comminution, but to directly decompress the spinal canal and affect the stabilization all in one setting.[2,4] The complex anatomy of this area—the pleura, diaphragm, retroperitoneal structures, and large vessels—make this approach somewhat less attractive to many surgeons,[10–12] and the morbidity can be substantial. The heart and the great vessels can limit access to the mid or upper thoracic spine, and the diaphragm can make it difficult to expose the entirety of the thoracolumbar junction. Postoperative pulmonary or gastrointestinal side effects can be substantial, especially in the elderly. The anterior approach is indicated for direct reduction of retropulsed fractured vertebral fragments in the setting of a neurologic deficit, severe comminution in the setting of short-segment posterior pedicle screw fixation, insufficient posterior elements for fusion, and the correction of severe kyphotic deformity secondary to malunion or failure of the posterior instrumentation.

CERVICAL STERNOTOMY

The lower cervical spine and upper two or three levels of the anterior thoracic spine can be accessed by performing cervical sternotomy (Fig. 29-6). A vertical incision is made anterior to the sternocleidomastoid and then continued distally over the
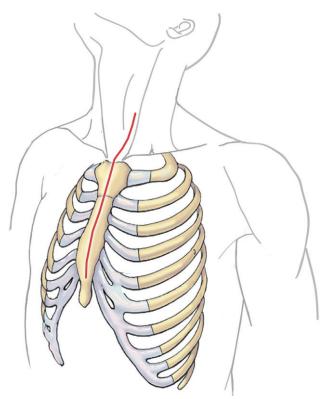
Fig. 29-6 Incision for a sternoclavicular approach to the upper thoracic spine. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437–1470.)

sternum. Usually, the brachiocephalic vein at the upper border of the manubrium is visualized and ligated. Once the sternum is divided with an oscillating saw, it is retracted, exposing the pericardium and costomediastinal pleural recesses. The esophagus and trachea are retracted, as is the common carotid artery, exposing the spine. A potential complication is that sternotomy is associated with significant risk of dehiscence and infection.

TRANSTHORACIC APPROACHES

The thoracic spine from T2 to T12 can be exposed via a transthoracic approach. Most surgeons prefer to approach the spine from a left-sided approach because manipulation of the aorta might be somewhat safer than that of the vena cava. If instrumentation is going to be placed along the lateral aspect of the spine, some surgeons prefer a right-sided approach rather than risk injury by the plates and screws to the pulsatile aorta.

The patient is placed on his or her side, preferably in a directly lateral position to aid in the placement of instrumentation (Fig. 29-7). The table can be broken to improve exposure or to correct fracture deformity. Intraoperative monitor-
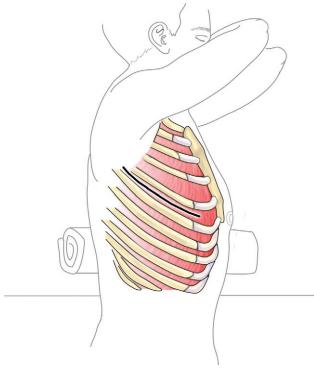

Fig. 29-7 Incision for a transthoracic approach to the mid thoracic spine. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437–1470.)

ing can be used but is not mandatory. Intraoperative C-arm might be helpful in identifying the injured level and verifying the placement of the transvertebral screws.

The incision usually is made parallel with the rib, approximately two levels superior to the vertebral body to be approached. A rib can be excised and used for reconstruction of the vertebral body, or the approach to the thorax can be intercostal by dividing the intercostal musculature. Division and release of the segmental vessels above and below the injured vertebra allow the great vessels to fall away from the spine (Fig. 29-8). The lateral aspect of the vertebral bodies should be exposed back to the junction of the rib head, which typically is removed. This facilitates access to the neuroforamen and helps define the posterior aspect of the vertebral body. The pleura and lung can be retracted with a malleable retractor, or anesthesia can provide selective one-lung (double lumen) ventilation.

RETROPLEURAL APPROACH

This is similar to the transthoracic transpleural approach except that it is generally performed only one rib proximal to the injured vertebra. The rib is first exposed subperiosteally and then the parietal pleura separated from the rib cage by
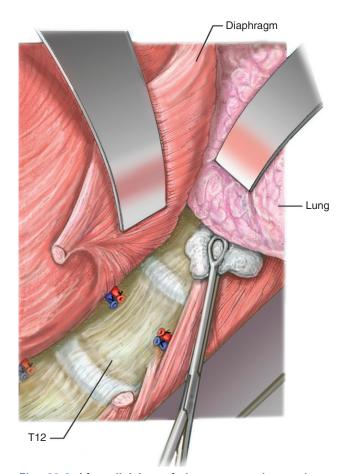
Fig. 29-8 After division of the segmental vessels at the mid-aspect of the vertebral bodies, blunt dissection is used to mobilize the lungs and great vessels. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437–1470.)
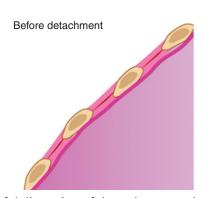
using fingers or swabs (Fig. 29-9). Resection of the head and neck of the rib can aid in the exposure. The intercostal vessels can be ligated and the nerve identified as a guide to the intervertebral foramen. Coagulation of the vessels near the foramen should be avoided to limit damage to the blood supply of the spinal cord.

THORACOSCOPY

Thoracoscopy more typically is used for the treatment of spinal deformity or the release of chronic posttraumatic deformity. Many authors,[13–15] however, have reported large series of minimally-invasive approaches to the anterolateral injured spine, not only for decompression but also for the placement of instrumentation and fracture reduction. Fractures at the thoracolumbar junction can have the diaphragm mobilized with this technique to expose the fracture for repair. Many can be treated with stand-alone thoracoscopic reconstruction and anterior instrumentation. A steep learning curve is involved, and the potential for severe complications is not insignificant; however, when compared with patients treated with open thoracotomy, the risk of pulmonary complications and the use of pain medications postoperatively seem to be less.[13,14]

ANTEROLATERAL APPROACHES TO THE THORACOLUMBAR SPINE

A transpleural, retroperitoneal approach provides access and allows close to full exposure from the upper thoracic to the lower lumbar spine. A retropleural approach typically allows visualization from the lower thoracic spine into the upper lumbar spine. With this approach, the diaphragm is incised not less than a centimeter from the chest wall.
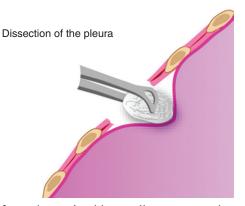

Fig. 29-9 Careful dissection of the pulmonary pleura away from the parietal layer allows a retropleural approach to the thoracic spine. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437–1470.)

The incision generally is centered over the 10th rib, which might be resected. Anteriorly, it is directed distally anterior to the anterior-superior iliac spine (Fig. 29-10). The retroperitoneum can be entered via the tip of the 11th or 12th ribs.

Mobilization of the parietal pleura is accomplished with blunt dissection anteriorly by using fingers or a sponge and elevating the pleura, diaphragm, and peritoneum (Fig. 29-11). For access to the lumbar spine, the crus of the diaphragm can be divided approximately 2 cm from the vertebral body. Segmental vessels can be divided, ligated, and/or clipped close to the aorta, allowing displacement of the great vessels. The psoas is mobilized by detaching it as far laterally as the pedicles to allow full exposure of the lumbar spine. Care must be taken to avoid injury to the

Fig. 29-11 Exposure of the thoracolumbar spine, with careful division of the diaphragm, typically 1 to 2 cm from its lateral attachment. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437-1470.)
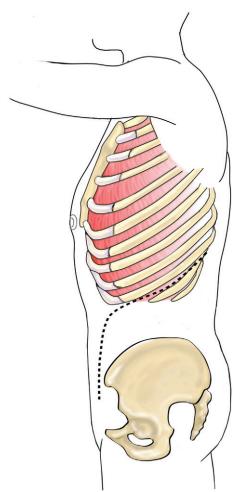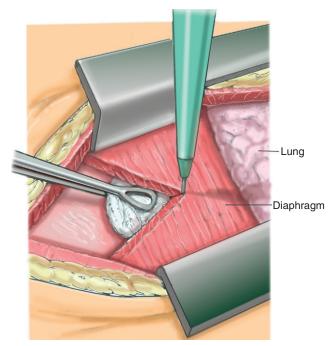

Fig. 29-10 Incision for a thoracoabdominal approach to the thoracolumbar spine. (Reproduced with permission from Kostuik J: Surgical approaches to the thoracic and thoracolumbar spine. In Frymoyer JW [ed]: The Adult Spine: Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1437–1470.)

genitofemoral nerve and the segmental vessels overlying the middle of the vertebral bodies.

SIMULTANEOUS ANTERIOR-POSTERIOR APPROACH

A simultaneous approach most commonly is indicated for the correction of late kyphotic posttraumatic deformities. It allows two teams of surgeons to simultaneously affect instrumentation and release and allows ultimate correction of the deformity without having to close and reposition the patient between stages. Typically, the patient is placed in the lateral position to allow the two surgical teams to work simultaneously; alternatively, the posterior instrumentation can be placed with the patient first in a more normal prone position and then turned to the lateral to allow the second anterolateral thoracoabdominal incision. Operative time, blood loss, and hospital stay can be significantly less than those with staged surgery.[7,16] Rarely, surgery for acute fracture can be performed in this manner, but with the advent of such rigid, short-segment instrumentation systems and the advancement of surgical techniques and approaches, it often is not indicated.

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Operative Techniques: Anterior Thoracolumbar Decompression, Fusion, and Instrumentation

INTRODUCTION

In 1931, Watson-Jones[1] reported his experience that "accurate reduction [of thoracolumbar fractures] was almost always possible, consolidation occurred without deformity, and provided that exercise in plaster was performed properly, functional results were excellent." Holdsworth[2] later reflected that "My experience with the treatment of vertebral fractures did not confirm this." In the ensuing decades, the natural history and management of thoracolumbar fractures has become better, yet incompletely, characterized. In parallel with this development, thoracolumbar surgical techniques have evolved, guided by the basic goals of restoration of spinal stability and protection of the neural elements.

Thoracolumbar fractures typically result from motor vehicle collisions (48% to 56%) or falls (19% to 36%).[3,4] They are often associated with non-contiguous spinal fractures (6% to 15%) or other injuries.[5,6] The high complexity of a typical trauma patient, in conjunction with a currently incomplete understanding of the management of thoracolumbar fractures, often leaves the treating physicians facing controversies related to operative indications, timing, surgical approaches, and techniques.[7] This chapter reviews the role for and application of anterior operative techniques used in the treatment of these fractures, including decompression, fusion, and instrumentation.

RATIONALE FOR ANTERIOR SURGICAL INTERVENTION

Thoracic and thoracolumbar spine fractures can be treated with anterior decompression and instrumented fusion. The biomechanics of the injury and the resultant stability need to be evaluated. Typically, two- or three-column injuries associated with instability and canal occlusion in patients with incomplete spinal cord injury may be considered for these techniques.

The anterior and middle vertebral columns typically bear compressive loads and are balanced by the annular and posterior ligamentous complexes. Interactions of anatomic elements normally allow for stable intervertebral motion. Functional instability often requires surgical intervention, but the concept of spinal stability may be elusive. White and Panjabi[8] defined clinical instability as the "loss of the spine's ability to maintain patterns of displacement under physiologic loads so that there is no major deformity or progression of deformity, no initial or additional neurologic deficit, and no incapacitating pain." This definition includes several parameters that are often used when considering surgery, namely abnormal motion or deformity, neurologic deficit, and pain. Biomechanical analysis of cadaver thoracolumbar burst fractures has demonstrated multidirectional instability.[9,10]

Thoracolumbar fracture classifications, described in an earlier chapter, have been developed to define specific injury patterns. Early classifications were based on morphologic description of injuries,[2,11–14] and later incorporated mechanisms of injury.[15,16] More recently, classifications have combined these parameters with treatment recommendations.[7] Surgery is generally advocated if inadequate results are anticipated from non-operative management.

Thoracolumbar fracture surgery can be performed using posterior, anterior, or combined approaches. For example, fractures addressed with an anterior approach may need posterior supplementation if there are noncontiguous injuries or a disruption of the posterior tension band. Many surgeons are most familiar with posterior approaches as these are performed more routinely. Such posterior operative techniques are covered in the following chapter. There are, however, compelling reasons to consider anterior decompression, reconstruction, and instrumentation, which are reviewed in the following sections.

Certain clinical and radiographic findings have been identified to provide rationale for anterior treatment. These include a large retropulsed fragment with canal compromise greater than two thirds, anterior comminution with kyphosis greater than 30 degrees, and time from injury of more than 4 days.[17] Anterior techniques are well applied to fractures that have poor reduction potential with a posterior approach. Decompression of anterior canal impingement may be limited from the posterior approach. Additionally, a kyphosis-reducing posterior construct must distract the anterior column, predisposing to anterior pseudarthrosis and the potential for ultimate construct failure.

Anterior techniques are most often applied in the acute or subacute setting, but they can be used to address delayed post-traumatic kyphosis that causes pain or neurologic deficit.[18,19] The use of late anterior techniques for revision after posterior-alone surgery has been evaluated. A series of 45 anterior decompressions performed for late pain and paralysis at an average of 4.5 years following fractures of the thoracolumbar spine demonstrated improved pain in 41 of 45 patients and improved neurologic function in 21 of the 25 patients with preoperative deficits.[20]

CONTRAINDICATIONS FOR ANTERIOR TREATMENT

Anterior decompression and stabilization of thoracolumbar fractures may be contraindicated by medical conditions that preclude the safe use of anterior surgical approaches; poor pulmonary function and obesity are both risk factors for complications of transthoracic surgery. Concomitant abdominal, diaphragmatic, and thoracic injuries often are associated with thoracolumbar spine injuries and need to be carefully assessed before planning anterior spinal surgery. Marked vertebral osteoporosis may also limit anterior stabilization options.

Anterior-alone constructs are most effectively balanced by an intact posterior liga mentous tension band. Biomechanical testing has demonstrated that if the posterior elements have been transected or if the posterior ligaments are not functional, anterior-alone instrumentation may not be sufficiently stable.[21] Thus, if there is deficiency of the posterior ligamentous complex, many surgeons will consider supplementing anterior constructs with posterior instrumentation. Eichholz et al.[22] performed a cadaveric biomechanical study comparing anterior-alone and posterior-alone constructs following L3 subtotal corpectomies. The anterior group received L3 wooden strut grafts with dual rod and screw instrumentation, and the posterior group received L1-L5 pedicle instrumentation. They found increased rigidity of the posterior instrumentation, which they attributed to the longer constructs.

Many thoracolumbar fractures, and particularly fracture-dislocations, cannot be managed with anterior-alone techniques. The successful reduction of such fractures typically requires posterior element manipulation or excision. Treating surgeons should also be cognizant of the fact that a neurologic deficit in patients with thoracolumbar burst fractures associated with a laminar fracture may be related to entrapped neural elements; in this case, a posterior approach should be considered.[23] If anterior approaches are necessary, however, combined approaches may be appropriate.

TIMING OF DECOMPRESSION AND STABILIZATION

The optimal time to provide surgical treatment is not well established. Progression of neurologic injury, however, which may be associated with increasing deformity, developing hematoma, or cord edema, is considered to be one indication for urgent surgical intervention. Many surgeons also consider neurologically incomplete patients a high priority to get to the operating room for early decompression of the neurologic elements. This is not well proven, however, and conflicting results have been reported in clinical studies.

A canine model of spinal cord injury has been used to demonstrate that the release of compression 1 hour after its application was associated with improved recovery as compared with compression more than 1 hour.[24] This would advocate expedited decompression of spinal cord compression.

On the other hand, Vaccaro et al.[25] found no significant difference between patients who underwent cervical spinal cord decompression at less than 72 hours after injury versus more than 5 days after injury in a prospective, randomized trial. A subsequent retrospective analysis of complications associated with cervical decompression before or after 72 hours following injury indicated no significant difference in complications.[26] It is difficult to tell if clinical differences were simply not able to be detected by these studies, if early enough time points were studied, or if, in fact, timing is not a crucial factor.

Carlson et al.[27] recorded canine regional blood flow and somatosensory evoked potentials after spinal cord compression, with release of compression after 5 minutes, and without release of compression. The results indicated that there is a very brief window for early decompression to allow optimal neurologic recovery. This may explain the reason for the varied results in human and animal trials; almost all "early" decompressions in humans were not achieved in this early narrow window. Additional clinical studies are underway to address the potential relationship between timing of decompression and neurologic outcomes.

Conversely, there may be advantages to delaying surgical intervention; complications of immediate surgery have been well described.[28] A delay will allow for full assessment of associated injuries, surgical planning, and assembly of the optimal surgical team. Specific to anterior approaches, a delay of surgery by a few days has been associated with lessened intraoperative bleeding.

In the acute setting, timing of decompression and stabilization typically requires coordination with trauma surgeons and other consultants. Concomitant injuries may take precedence, including acute intracranial, abdominal and thoracic injuries, unstable pelvic fractures, and open long-bone fractures. Most surgeons recognize that the presence of such injuries may increase the risk of complications of immediate surgery and thus may delay surgical treatment. It has recently been demonstrated, however, that after a thorough trauma evaluation, early surgery (<72 hours) is associated with fewer complications, shorter hospital and *SICU* stays, and less ventilator support in severely injured trauma patients.[29] It is imperative, however, that no critical steps of the modern trauma assessment and acute care are supplanted by an operative intervention.

PREOPERATIVE PLANNING

Preoperative planning is vital to successful treatment. Desired decompression and reconstruction goals must be established. CT images can be used to evaluate surrounding structures as well as bony detail, including anatomic landmarks and dimensions for reconstructive planning. MRI can ascertain the status of the relevant ligamentous structures and neural element compression. Specialized equipment and consultants must be in place. Neurophysiologic monitoring is often used.

OPEN ANTERIOR THORACOLUMBAR DECOMPRESSION

Anterior surgical approaches offer excellent access for direct decompression of the neural elements. Some degree of decompression may be performed from the posterior, either indirectly through the use of ligamentotaxis or directly by the manipulation of fracture fragments. Some authors have found anterior decompression to result in greater neurologic recovery as compared with posterior-alone management,[30–32] but others have not found significant differences.[4,33] It is clear, however, that posterior decompressions, whether indirect or direct, may not be achievable to the degree that can be obtained from an anterior approach to address retropulsed fragments. This is particularly true at the cord level, where lesser manipulation of the neural elements is possible.

The anterior approach to the thoracolumbar spine, originally described in 1975 by Paul et al.[34] as a transthoracic approach for decompression of acute spinal cord injuries, has been well applied to the entire length of the spine. The success and acceptance of these anterior approaches is due in part to the greater ease of a complete decompression as compared with posterior-alone surgical techniques. Optimal visualization of the anterior thecal sac can be accomplished after corpectomy. These factors may not be important for neuro-

logically intact or complete patients, but become a significant consideration for patients with incomplete spinal cord injuries.

In performing a decompression, the spine is exposed one level above and below the damaged segment. The segmental vessels at all three levels are identified, isolated, and ligated over the anterior third of the vertebral body so as to not interfere with collateral flow to the cord (Figs. 30-1, *A* to *D*). It can be useful to temporarily clamp segmental vessels and evaluate for electrophysiologic monitoring changes before vessel ligation. The periosteum is elevated posteriorly along the pedicles to place a blunt instrument within the neuroforamen to help establish anatomic landmarks and retract the soft tissues. This allows accurate delineation of the margins of the spinal canal.

The disks cephalad and caudal to the damaged segment are identified and excised. The corpectomy is then performed, which is often facilitated by the fracture itself. A pedicle may be removed to gain access to the lateral border of the spinal canal. A diskectomy can then be accomplished back to the level of the annulus and posterior longitudinal ligament (PLL). Removed bone can be saved as autologous graft.

The anterior longitudinal ligament (ALL) is left intact with an anterior rim of bone to preserve stability. Care should be taken to remove bone as far as the contralateral pedicle to achieve an adequate decompression (Fig. 30-2). After decompression, the PLL ligament should bulge anteriorly in a uniform fashion from one endplate to the next, and from pedicle to pedicle. If not, removal of the ligament may be required to identify remaining disk or bone encroaching on the spinal canal.

ENDOSCOPIC ANTERIOR THORACOLUMBAR DECOMPRESSION

Endoscopic anterior spinal decompression has been used in the management of thoracolumbar trauma. Authors have reported a reduction in complications as compared with more traditional anterior decompression techniques, while achieving excellent canal clearance. Beisse et al.[35] reported a series of 30 patients who had undergone anterior endoscopic decompression with interbody reconstruction. Transdiaphragmatic thoracoscopic and endoscopic retroperitoneal surgical techniques have also been performed in the treatment of fractures at the thoracolumbar junction, permitting decompression and instrumented reconstruction.[36]

Patient positioning and trocar placement are critical. Several different transdiaphragmatic approaches to the thoracolumbar junction have been described, using retraction of the diaphragm or minimal detachment to facilitate access. Endoscopic retroperitoneal approaches are also described to address the thoracolumbar junction.
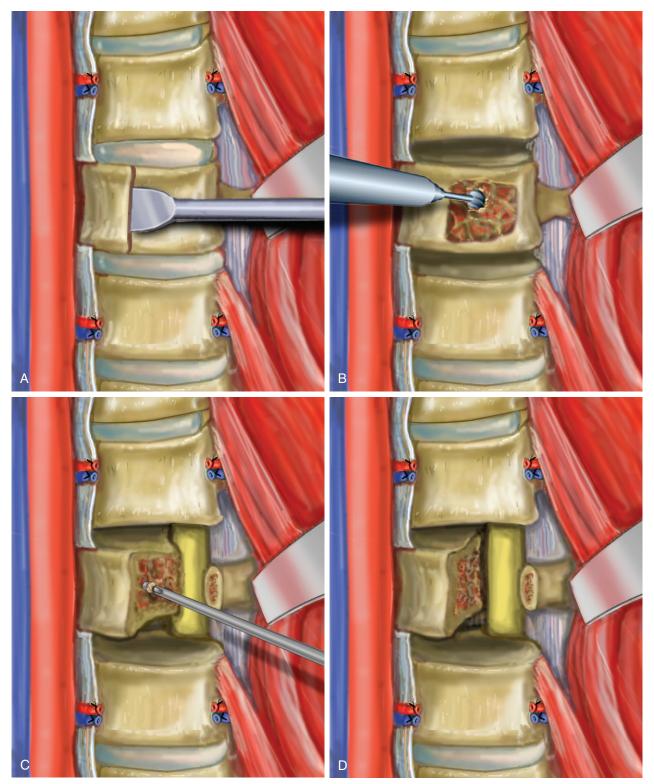
Fig. 30-1 Corpectomy is performed. *A,* After exposure and ligation of segmental vessels, the vertebral body is osteotomized and kept as a graft. *B,* Dural decompression with a burr and rongeur. *C,* Bone taken from the contralateral side. *D,* Extent of decompression.
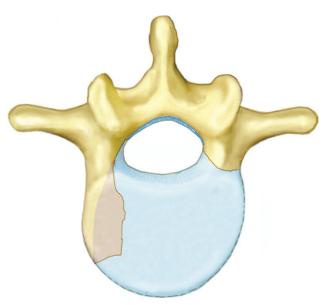
Fig. 30-2 Following decompression, the area of bone resection should extend as far as the contralateral pedicle. Uniform bulging of the posterior longitudinal ligament (PLL) provides confirmation.

Following fluoroscopic identification of the target vertebra and placement of trocars, the cephalad and caudal disk interspaces are exposed. At the thoracolumbar junction, the diaphragmatic insertion on the spine may have to be taken down. Once exposed, techniques similar to their open counterparts can be used.

ANTERIOR THORACOLUMBAR RECONSTRUCTION

Biomechanically, an advantage of anterior thoracolumbar fracture management is the ability to perform reconstruction of the anterior column. This is highlighted by the load-sharing concept introduced by McCormack et al.[37] Their retrospective review of 28 patients with three-column injuries, fixed posteriorly with screw and rod constructs, was used to identify injury characteristics that would predict failure of this fixation method. A nine-point system for predicting failure of posterior-alone fixation was thus developed, with one to three points assigned to each of three factors: extent of comminution, fragment displacement, and kyphosis correction. The authors suggested that anterior reconstruction is required if the anterior column cannot load-share; point totals over 6 suggested obligatory anterior reconstruction. Parker et al.[38] subsequently reported their results using the load-sharing classification; those patients with a high degree of comminution, displacement, and kyphosis did have a better result with anterior as compared with posterior stabilization.

A number of different types of struts can be used for reconstruction of the anterior column. Although tricortical iliac crest autograft, which supplies live osteogenic progenitor cells, resident growth factors, and a histocompatible structural scaffolding, is the material most preferred for promoting fusion, the morbidity associated with the harvest of a graft of this size is significant.[39,40] As an alternative, allografts are commonly used, including tibial, humeral, and others. Ribs removed during the approach can be used, as well, but offer relatively poor resistance to compression.

Commonly, a synthetic strut composed of peak, carbon fiber, or metal is used. Expandable cages are also being used with increasing frequency. These grafts provide immediate mechanical support against compression while bony incorporation ensues. The use of these interbody devices can preclude the harvest of iliac crest autograft and can be packed with local autograft harvested by anterior vertebral decompression or rib resection. Cancellous allograft, demineralized bone matrix, and bone morphogenetic proteins may also be used in conjunction, both packed within and around the device.

In many clinical trauma scenarios, the differences among implants may not be of great significance. The primary goals of resistance to compression and rotation are achieved by many constructs, any of which can lead to successful fusion. (Figs. 30-3, *A* to *C*). Nonetheless, there are considerations that come into play. For example, the relatively soft bone of the elderly patient may be more likely to allow subsidence of rigid metallic implants than allografts, which better match the local modulus of elasticity. Also, differences in bone graft surface area may allow different degrees of bony ingrowth. Further, expandable cages are challenging to get good graft fill in their expanded form.

Polymethylmethacrylate (PMMA) can also be considered as a graft alternative. It has the advantage of immediate stability obtained through intercalation of the material with native bone interstices. It has excellent strength in compression, and the ability to be used in conjunction with pins and wires (Fig. 30-4). Antibiotics may be mixed with the cement and may reduce the risk of infection. During its exothermic curing phase the neural tissues are at risk for thermal injury and must be protected. In general, however, PMMA is not used in the young population that most commonly sustain thoracolumbar fractures.

Reduction is often facilitated by patient positioning. A reduction maneuver can be performed by placing a spreader/distractor into the corpectomy site and/or by manually applying an anterior force on the posterior elements to reduce the kyphosis. After the dimensions of the desired graft or interbody device are determined, the strut is measured and tamped into position, under direct visualization. Care is taken to ensure that the implant does not encroach on adjacent structures such as the dura and blood vessels. Once the graft or device is in position, vertebral distraction is released. One should ensure that the graft is large enough, both to adequately reduce kyphosis and to reduce the risk of extrusion. When significant kyphotic deformity exists, there may be mechanical benefit to placing the graft more anterior.
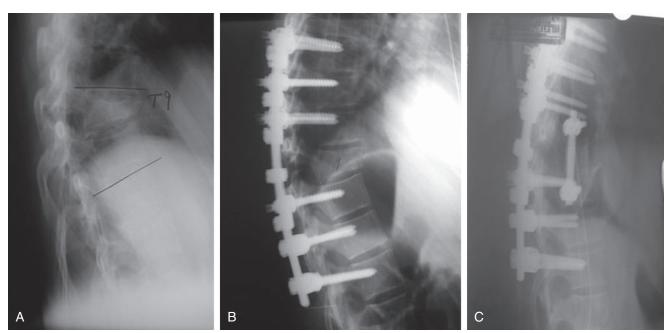
Fig. 30-3 *A* to *C,* Case of a 31 year old who sustained a T9 fracture after being thrown from a horse. There were no neurologic deficits. She was initially treated with a posterior approach and instrumented fusion with a pedicle screw-rod construct from T6 to T12. There were concerns regarding anterior column support and reduction of postoperative kyphosis. She was therefore revised with an anterior T9 corpectomy, allograft placement, and stabilization with a single-rod construct. *A,* Injury radiographs. *B,* Initial surgical procedure. *C,* Revision.

The ultimate goal of long-term spinal stability depends on the success of the fusion procedure. Although implants provide immediate stability, meticulous fusion techniques must be used to achieve a biologic fusion. Vertebral endplates are prepared by removing cartilaginous tissue to the level of finely bleeding bone, but care is taken to preserve the subchondral bone to reduce the risk of graft subsidence.[41] Cancellous autograft, harvested with the decompression, can be packed within the allograft strut, cage, or expandable device, and or packed around the intervertebral graft. When the grafting is complete, there should be adequate space between the neural elements and the graft.

ANTERIOR THORACOLUMBAR INSTRUMENTATION

Anterior instrumentation is commonly used in combination with anterior column strut reconstruction. In the past, stabilization may have been accomplished with staged posterior instrumentation after anterior decompression and strut grafting.[42] Modern anterior devices, however, such as plate/screw and rod/screw constructs have allowed for excellent stability to be obtained with anterior-alone instrumentation. And when anterior instrumentation is used in conjunction with posterior instrumentation, shorter posterior constructs may be used.[43]

An animal corpectomy model has been used to observe the biomechanics of thoracolumbar fracture. Gurr et al.[44]

demonstrated that although the strength in flexion, rotation, and axial loading was improved with the addition of iliac crest graft to the corpectomy, it still allowed three times the displacement in compression and demonstrated less than one third the torsional stiffness of the native, intact spine.

The origins of anterior spinal instrumentation are with the surgical treatment of scoliosis; the Zeilke rod was prototypical of early designs. With a single rod, however, the Zeilke device demonstrated suboptimal stiffness in bending and torsion.[45] The subsequent development of the dual-rod Dunn fixation system represented a structural improvement, but had significant shortcomings related to its high profile design and ventral position, risking erosion of the aorta.[46] In 1984, the Kaneda system was introduced.[47,48] Biomechanical testing determined instrumentation rigidity to be greater than native spines in a bovine model[49] and long-term clinical results have been excellent.[50] Single-and double-plating systems have since been developed, with still greater rigidity.[51]

Generally, double rod or plate constructs are used for stand-alone anterior reconstructions. If posterior instrumentation is planned, then a single-rod construct may be considered. The goal of the anterior devices is to afford immediate stability, prevent graft extrusion, and limit settling.

Preparation of the vertebral bone for fixation includes removal of osteophytes, endplate prominences, and rib heads, to create a relatively flat surface. The device should be fixed
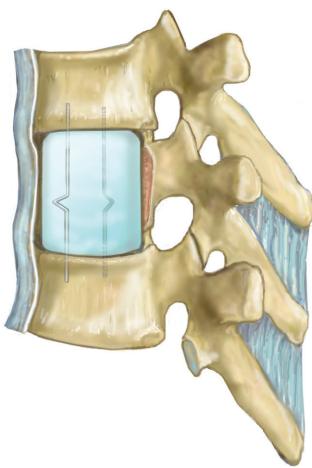
Fig. 30-4 Polymethylmethacrylate (PMMA) used as a graft alternative. Immediate stability is conferred through intercalation of the material with native bone interstices.

posteriorly to avoid contact with the adjacent vessels, but not be posterior enough to impinge on the neural elements. Screws are generally placed bicortically (Figs. 30-5, *A* and *B*). Care must be taken to limit the screw length to prevent immediate or late vascular injury on the far side of the vertebral body. Also, care must be taken to avoid the adjacent disk spaces.

After the device is in place and after applying compression across the segment, thereby incarcerating the interposed graft or device, the construct is locked into place. In the case of severe osteoporosis or loss of endplate integrity, supplementation with posterior instrumentation may be warranted.

POSTOPERATIVE CARE

A total contact thoracolumbar orthosis is typically used postoperatively for 3 months. Patients are encouraged to ambulate and participate in activities of daily living as soon as they are able. Patients with spinal cord injury are managed accordingly in conjunction with a multidisciplinary spinal cord injury rehabilitation team.
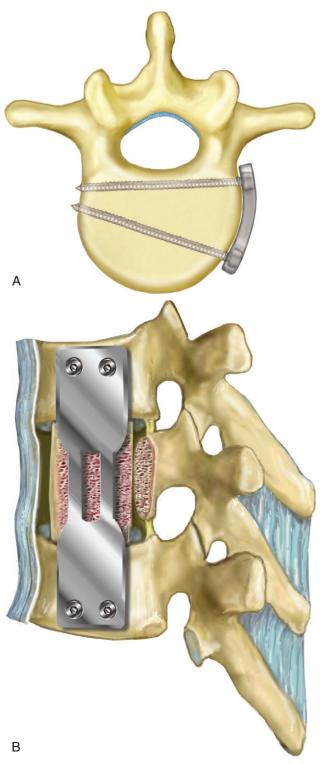

A

B

Fig. 30-5 *A,* Thoracic screws through a rod or plate construct should be placed bicortically with care taken to limit screw length. *B,* Plate using bicortical screws with an interposed iliac crest bone graft.

PITFALLS AND SALVAGE OPTIONS

Complications from an anterior approach to the thoracolumbar spine may include anterior dural tear, injury to the neural elements, great vessel injury, and excessive bleeding. Precautions to avoid these complications include the use of an experienced surgical team familiar with anterior spinal exposures, the routine use of neurophysiologic monitoring, and intraoperative autotransfusion.

The complications related to this procedure include those intrinsically related the approach itself. Additional technical challenges may include severe bleeding from the epidural plexus during decompression, making meticulous and immediate attention to epidural bleeding of paramount importance. The decompression should proceed so as to pull the compressive burst fragments away from the compromised neural elements. This will minimize the chances of further compression from the instruments during the course of the procedure.

RESULTS OF ANTERIOR TECHNIQUES

A number of studies have presented the results of anterior decompression and fusion. Many of these have reported encouraging outcomes. Kostuik[18] described 80 thoracolumbar fracture patients with an average postoperative recovery of 1.6 Frankel grades. McAfee et al.[32] also described clinically significant neurologic improvement after anterior decompression and stabilization in 37 of 42 patients 2 months after thoracolumbar fractures with incomplete neurologic deficits. Nearly half of the patients whose quadriceps and hamstrings were too weak to permit walking regained full independent ambulation. One third of the patients with conus injury had bladder function recovery. Other studies also report that restoration of bladder function may be more effective after anterior decompression.[32,33]

More recently, Sasso et al.[52] reported a series of 40 patients who underwent anterior-only stabilization of three-column thoracolumbar injuries with a mean canal compromise of 68.5% and a mean vertebral kyphosis of 44.5 degrees. Neurologic improvement of at least one Frankel grade was seen in 91% of patients. Mean segmental kyphosis was improved, with a range from 22 degrees to 7 degrees. They concluded that stand-alone anterior techniques were sufficient for the treatment of three-column thoracolumbar fractures.

Wood et al.[53] performed a multicenter, prospective randomized study of anterior versus posterior treatment of stable single-level burst fractures at the thoracolumbar junction (T10-L2) in 38 patients without neurologic deficits. Those randomized to anterior treatment underwent subtotal corpectomy, allograft, and local bone reconstruction with instrumentation. Those randomized to the posterior group were treated with a three- or four-level screw and hook construct with iliac crest bone graft. The anterior-alone group had fewer complications and required fewer subsequent surgeries than the posterior group.

Others have found less encouraging results. A multinational retrospective review of 1019 fractures reported that anterior surgery was not more effective than posterior surgery in improving the neurologic function, as assessed by the Frankel or Motor Index scales. Anterior surgery was found to be statistically more effective, however, as assessed by the Manabe scale. Their data also indicated that anterior surgery was more beneficial in improving bladder dysfunction compared with posterior surgery.[4]

CONCLUSION

A stand-alone anterior procedure is best indicated in the setting of good bone quality at the thoracolumbar junction in a patient with an intact posterior osteo-ligamentous complex. An understanding of the biomechanics of the thoracolumbar injury will assist the surgeon in determining if anterior techniques, either alone or together with posterior treatment methods, will be appropriate for each patient. Anterior approaches to the thoracic spine can provide a real advantage in facilitating decompression and in kyphosis correction with anterior stabilization. Surgical outcomes for thoracolumbar injuries are likely related to the success of the decompression, stabilization, and fusion procedures. This may prove technically difficult, although attention should be directed toward achieving these ends. The management of complex thoracolumbar fractures is in evolution, as it was decades ago when Watson-Jones and Holdsworth presented their perspectives; clinical cohort studies are currently underway to help guide the ever-improving care of these challenging injuries.

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C H A P T E R

31

CHRISTOPHER M. BONO,

JOHN D. PRYOR

Operative Techniques: Posterior Thoracolumbar Decompression, Fusion, and Instrumentation

INTRODUCTION

Nearly 90% of fractures of the spine occur in the thoracic or lumbar region.[1] The majority occur between T11 and L2, commonly referred to as the thoracolumbar junction.[2] Since the first attempts made at classification in the 1940s, advancements in imaging have contributed to the understanding of the pathomechanics of these injuries. This, in turn, has stimulated an evolutionary discussion of thoracolumbar injury classification within which disagreement still persists concerning the optimal system.[3–6] Despite this unclarity, a number of thoracolumbar injury patterns have been identified and seem to be consistently reported in the literature.

INJURY TYPES

COMPRESSION FRACTURES

Compression fractures are wedge fractures usually isolated to the anterior and middle aspects of the vertebral body. The posterior vertebral body is not involved, in contrast to burst fractures. This fracture pattern is usually considered stable; however, posterior ligamentous complex (PLC) disruption may be associated with more severe injuries indicating instability.

BURST FRACTURES

Comminution of the vertebral body that includes the posterior cortex is characteristic of a burst fracture. Although not always present, it most often demonstrates retropulsion of fracture fragments into the spinal canal. With burst fractures, the PLC may or may not remain intact. Contradistinct to fracture-dislocations, which may also be associated with comminuted vertebral body fractures, translational deformity is not present with burst fractures.

FLEXION-DISTRACTION INJURIES

Also known as Chance or seatbelt injuries, this pattern is characterized by tensile failure of the posterior and anterior elements, either through the bone or ligaments or a combination of the two. These injuries are classically described as the result of a flexion moment around an axis of rotation anterior to the spinal column. Often, a component of vertebral body compression is present, suggesting the mechanism is not always pure flexion-distraction.

FRACTURE-DISLOCATIONS

The distinctive attribute of fracture-dislocations is translational deformity or listhesis in the sagittal and/or coronal planes. These markedly unstable injuries are typically the result of high-energy trauma and may variably demonstrate fractures of the anterior and posterior elements. Fracture-dislocations of the thoracolumbar spine are associated with a very high rate of complete neurologic deficit.[7,8]

EPIDEMIOLOGY

Reports of neurologic deficit from spinal column trauma date to nearly 5 millennia ago as recorded in the Edwin Smith Surgical Papyrus. Fifteen percent to 20% of thoracolumbar fractures result in neurologic deficit, affecting approximately 1 in 20,000 residents of the United States.[4,9] Modern living, replete with motorized vehicles and other potential high-energy injury sources, has yielded a substantial

increase in the incidence of thoracolumbar fractures. As with trauma in general, young men are the most often injured group.[2] Blunt trauma, by motor vehicle accident, fall, or crush, is the primary mode of injury. Motorcycle accidents are associated with a greater chance of more severe spinal column trauma than car accidents.[10] Gunshot wounds to the spine are an increasingly *occurring* cause of thoracolumbar injuries;[11] underlying osteoporosis is an increasingly *recognized* causative factor.[12]

CLINICAL PRESENTATION

Injuries of the thoracolumbar spine often present in the setting of a polytraumatic event. A common exception includes low-energy compression fractures in osteoporotic patients. Initial assessment and clinical management of the polytraumatized patient includes that outlined by the Advanced Trauma Life Support (ATLS) protocol. Neurogenic shock may be present, which must be distinguished from hypovolemic shock. A so-called seat belt sign, noted by a band of ecchymosis across the chest or abdomen, may suggest a flexion-distraction injury, which often has a concomitant hollow viscus injury. A high suspicion must be maintained for spinal injuries, particularly with concomitant distracting injuries that compromise the reliability of the initial physical examination. In one study, thoracolumbar injuries were initially missed in 24% of polytrauma patients.[13]

In the alert, cooperative patient, a thorough neurologic examination includes assessment of motor strength, touch/pain/temperature sensation, perianal sensitivity, rectal tone, and bulbocavernosus reflex. Many elements of this exam may not be initially possible because of a patient's obtunded mental status or other life-threatening injuries. The patient should be log rolled after a baseline neurologic examination. Focal tenderness, ecchymosis, open wounds, bogginess, or other posterior soft tissue defects may be encountered in addition to malalignment or palpable step off of the spinous processes.

DIAGNOSTIC METHODS

With the presence of a concerning or unreliable examination, anteroposterior (AP) and lateral radiographs of the thoracic and lumbar spine or computed tomography (CT) with sagittal and coronal reconstructions should be obtained.[14] The AP film may reveal changes in the coronal alignment, interpedicular distance, or interspinous process distance. Coronal malalignment suggests mechanical instability as a result of high-energy trauma. More than 2.5 mm of translation in the sagittal or coronal planes is highly suggestive of gross discoligamentous failure and instability.[15] Lateral displacement of fracture fragments is signified by an increased distance between the pedicles of the injured level relative to adjacent levels. Interspinous process distance is used to indirectly assess the integrity of the PLC.[16,17]

Sagittal alignment should be scrutinized on the lateral view. The Cobb method can be used to quantify kyphosis by measuring the angle formed at the intersection of a line drawn along the superior and inferior endplates of the vertebrae cranial and caudal to the injured segment, respectively.[18] PLC disruption is likely when kyphosis of an injured segment exceeds 30 degrees.[19–24] Vertebral body height loss should be expressed as a percentage of the height of the adjacent, uninjured vertebra. Biomechanical data suggest 50% loss of vertebral body height is indicative of posterior instability.[25] The anterior and posterior vertebral body lines can be used to assess translation, the former being less reliable because of the frequent presence of degenerative changes.

Helical CT scans are more efficiently obtained and provide greater detail of vertebral body fragments and posterior osseous structures than plain radiographs.[26] Vertebral body comminution, whose presence may directly affect treatment, may be revealed.[27–30] Furthermore, plain radiographs have been shown to underestimate the amount of canal compromise by 20%.[31] Notably, axial CT images alone may not adequately reveal translational deformity, highlighting the importance of sagittal and coronal reconstructions.

Magnetic resonance imaging (MRI) provides better visualization of soft tissues and the neural elements. Conditions that may be missed by other modalities, such as disk herniations, epidural hematomas, PLC disruption, or spinal cord edema, are easily detected by MRI. It is particularly useful when evaluating a neurologic deficit that does not correspond with plain radiographic or CT findings.

CLINICAL MANAGEMENT

DIAGNOSTIC TESTS

Following initial evaluation and imaging protocol, thoracolumbar fractures can be classified into one of the four aforementioned descriptive categories. Compression fractures are those in which only the anterior and middle of the vertebral body is compromised. Stability is dependent on the integrity of the PLC, which can be evaluated with radiographic measurements of vertebral height loss and focal segmental kyphosis. PLC disruption is strongly suggested if vertebral body height loss exceeds 50%, kyphotic deformity exceeds 30 degrees, or lateral radiographs reveal interspinous widening. In equivocal cases, MRI can be used for PLC assessment.

If comminution of the vertebral body and fracture of the posterior cortex is present, there is a bursting component to the injury. Retropulsion of one or more fracture fragments is usually seen. As in compression fractures, radiographic indicators of PLC competence must be assessed to determine stability. Other radiographic findings of a burst fracture include widening of the interpedicular distance. Compromise of the

spinal canal can be gauged as a percentage of the total canal diameter and has been used by some in surgical decision making.[32,33] However, little evidence exists that establishes a threshold for canal compromise beyond which surgical intervention is indicated in the setting of an intact PLC and no neurologic deficit.[34–36]

NONOPERATIVE APPROACHES

Nonoperative treatment usually entails external immobilization. Hyperextension devices, such as a Jewett brace, can resist sagittal flexion forces. These braces offer minimal counteraction to rotation or lateral flexion. Custom-fit thoracolumbar sacral orthoses (TLSO) provide support in multiple planes and are more appropriate for nonoperative treatment of burst fractures. Most braces have been shown to increase motion across the lumbosacral junction. For fractures above T5, a cervical extension should be used. A rotating bed may help reduce the complications of recum-

bency in patients with thoracolumbar injuries who require strict bed rest.[37]

INDICATIONS FOR POSTERIOR SURGERY

Although a number of general principles applies, the indications for posterior surgery in the treatment of thoracolumbar injuries can be considered in terms of the specific injury patterns (Fig. 31-1).

COMPRESSION FRACTURES

Most compression fractures are inherently stable and can be managed nonoperatively. Patients in whom no neurologic deficit is present and whose fractures exhibit minimal vertebral body height loss (less than 10%) can be safely mobilized. Fractures resulting in less than 30% or 40% of vertebral body height loss and less than 20 to 25 degrees of kyphosis are usually considered stable and can be managed with a Jewett hyperextension brace for 6 to 8 weeks. Posterior stabilization
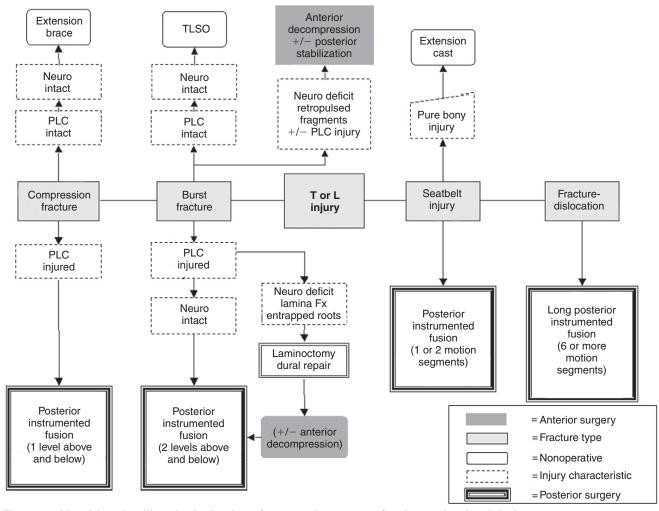

Fig. 31-1 Algorithm detailing the indications for posterior surgery for thoracolumbar injuries.

is usually recommended when there is radiographic evidence of PLC disruption.[19–25,38]

BURST FRACTURES

As with compression fractures, the criterion of an intact PLC serves as the basis for nonoperative management in a patient without neurologic deficit. Two prospective studies demonstrated similar outcomes between operative and nonoperative management of patients in this clinical scenario.[39,40] Others have found neurologic deterioration in patients who have sustained injuries compromising more than 50% of the canal.[32] It is unclear if this cohort also had PLC disruption, however. There exists little evidence on which one can establish a threshold canal compromise beyond which operative management is indicated.[32–36] Patients with a neurologic deficit may rarely warrant nonoperative treatment if they are not medically suitable to undergo invasive procedures.

The presence of a neurologic deficit, PLC injury, or progressive kyphosis are relative indications for posterior surgical stabilization. Most surgeons agree that a burst fracture in a patient with neurologic deficit indicates surgery regardless of radiographic indicators of mechanical stability. Progressive neurologic deficit is a strong indication for urgent stabilization and/or decompression. Improved mobility for the care of a polytraumatized patient in whom bracing is not feasible is also an important relative indication for surgical stabilization.

The goals of posterior surgery for burst fractures are primarily sagittal realignment and stabilization more so than decompression. We recommend posterior stabilization and fusion for neurologically intact patients with unstable burst fractures (i.e., PLC disruption). For patients with neural deficit, some have reported the use of laminectomy for decompression with posterior stabilization to be effective.[41] Laminectomy alone would lead to additional posterior instability and therefore should be used only in combination with instrumented stabilization and fusion. Posterior stabilization alone may be used in patients with thoracic level injuries who have a truly complete spinal cord injury as this subset of patients is unlikely to have useful neurologic return. Posterior stabilization alone may also be used in those patients with a neurologic deficit with little or no canal compromise (Fig. 31-2). In our practice,
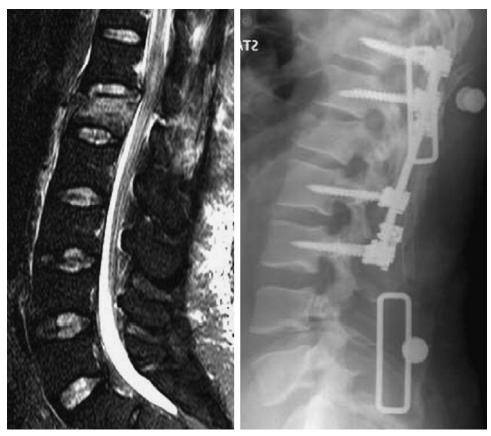

Fig. 31-2 In some cases, neural injury with burst fractures can be associated with little or no canal compromise. The mechanism of neural injury is likely distractive or a brief compression that quickly recoils. Formal decompression, either anterior or posterior, is unlikely to benefit neural recovery. In the MRI *(left)* of a 48-year-old man, an unstable burst fracture is seen because there is discontinuity of the PLC. However, there is minimal fragment retropulsion, with no mass effect on the conus medullaris. This patient was treated with a posterior stabilization and fusion without laminectomy or anterior decompression *(right).*

however, a burst fracture with neurologic compromise is usually treated by an anterior decompression and stabilization with or without posterior stabilization. Exceptions include burst injuries of T3 or T4, which are difficult to approach anteriorly, in a patient with an incomplete spinal cord injury. Direct decompression through an all-posterior approach can be an option in this instance.

FLEXION-DISTRACTION INJURIES

Flexion-distraction injuries are unstable by definition; thus, surgical intervention is typically indicated. A possible exception is that of a purely osseous injury that reduces well in an extension brace. Successful outcomes with this approach have been shown when the initial kyphotic deformity is less than 15 degrees.[42] Because of the mechanism of spinal cord injury usually being that of distraction rather than compression by retropulsed fracture fragments, formal decompression in the setting of a neurologic deficit is not common; stabilization is thought to offer the greatest benefit by decreasing the chance of further neural injury. Retropulsed fragments, a herniated disk, or a hematoma may be the source of neurologic insult, however, and should be decompressed surgically through an appropriate approach.[43]

The primary goal of surgery for flexion-distraction injuries is restoration of alignment and stability. In most cases the anterior longitudinal ligament (ALL) and portions of the anterior annulus remain intact. This makes posterior stabilization techniques particularly well-suited for such injuries. Posterior instrumentation and fusion of a single level can be adequate in some cases, provided the pedicles at the levels intended to be instrumented are intact and in continuity with the vertebral bodies (Fig. 31-3).

FRACTURE-DISLOCATIONS

As stated previously, thoracolumbar fracture-dislocations are the result of high-energy trauma that yields a markedly unstable spine. Surgical stabilization is generally indicated for these injures because nonoperative treatment would require postural reduction followed by a prolonged term of bracing and bed rest. Canal compromise from fracture-dislocations is usually the result of vertebral misalignment. As such, decompression is primarily achieved by realigning the spine, which is best done through long-segment posterior stabilization.

OPERATIVE PROCEDURE AND TECHNIQUE

OPERATING ROOM SETUP

Following intubation, the patient should be carefully log rolled into the prone position on a padded spinal frame, such as a Jackson table. Knees should be flexed to relieve any tension on the sciatic nerve. Allowing the abdomen to hang free decreases intra-abdominal pressure that helps collapse the epidural venous plexus and reduce blood loss. The skin is
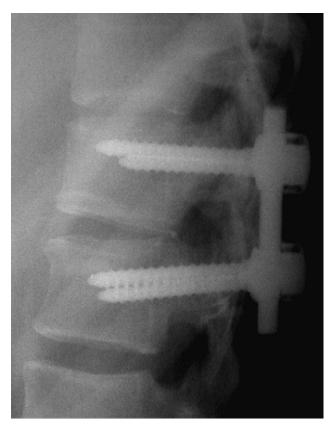

Fig. 31-3 Seatbelt or Chance injuries can usually be managed with short-segment posterior fixation. In the lateral radiograph shown, a young woman with a purely ligamentous flexion-distraction injury was treated with fusion and instrumentation of one motion segment (two vertebrae).

then prepared and the patient draped in the usual sterile fashion. The iliac crests should be included in the skin preparation when autologous bone grafting is planned.

Fluoroscopic imaging equipment should be available with appropriate technical staff. Adequate radiographic visualization should be verified before proceeding. The chance for a successful, uncomplicated procedure is optimized when the details and nuances of the devices are known to both surgeon and operating room staff. Some recommend somatosensory-evoked potential monitoring in neurologically intact or incomplete spinal cord injuries. This provides physiologic data during screw insertion and reduction maneuvers.

OPERATIVE TECHNIQUE: POSTERIOR STABILIZATION AND FUSION

EXPOSURE

A posterior midline incision is made spanning the appropriate levels. This is carried down with a scalpel to the subcutaneous fascia. Electrocautery is then used to dissect down to the deep

fascia. Continuing with electrocautery, the fascia is incised on either side of the spinous processes. Subperiosteal dissection is taken out and over the facet joints for a complete exposure of the posterior aspect of the levels to be operated. Transverse processes are exposed to their end in the lumbar spine or the costovertebral junction in the thoracic spine. The facet joints are visualized and opened using a small curette.

PEDICLE SCREW INSERTION

A small burr is used to mark starting holes for pedicle screws. In the thoracic spine, the starting point is at the base of the superior articular process, just medial to the superomedial portion of the transverse process (Fig. 31-4). In the lumbar spine, the starting point is at the intersection of a transverse line through the mid-aspect of the transverse process and longitudinal line just lateral to the mid-aspect of the facet joint (Fig. 31-5). In our practice, this starting point is marked using both AP and lateral fluoroscopic views. On the AP view, the burr is used to mark a point just lateral to the halo of the pedicle. On the lateral view, the burr hole is aligned with a trajectory along the anatomical axis of the pedicle.

With the starting point confirmed to be in good position, the burr is advanced into the bone approximately 3 to 5 mm. Next, a pedicle finder is inserted into the starting hole. It is angled toward the midline about 10 to 30 degrees, depending on the level b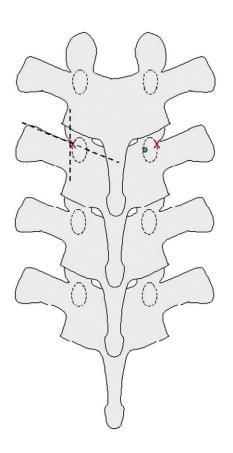eing instrumented. Ensuring that it is aligned with the pedicle on the lateral view, it is advanced into the vertebral body. To avoid medial penetration of the pedicle cortex, the pedicle finder tip should not extend beyond the medial border of the pedicle halo on the AP view before arriving at the posterior vertebral body on the lateral view (see Fig. 31-5).

The pedicle finder can be used to measure the optimal length of the screw. After it is removed, a ball-tip probe is inserted to sound the pedicle hole margins. The hole may be tapped if desired. It is our practice to undertap by 1 mm in most cases. In cases in which a screw path must be repositioned, tapping line-to-line (i.e., the same size as the screw) can be helpful. The screw is then inserted until purchase is maximized. The final screw position is checked using fluoroscopy.

If a thoracic pedicle is too small to accept the smallest screw, a lateral extrapedicular insertion technique may be used with equivalent biomechanical purchase. The starting point is more lateral than typical transpedicular insertion (Fig. 31-6). In addition, the medial angulation is more exaggerated to avoid lateral penetration through the vertebral body.

HOOK PLACEMENT

Although it is our practice to use pedicle screws whenever possible, many surgeons prefer to use hooks in the middle and upper thoracic regions. Pedicles in these regions can be
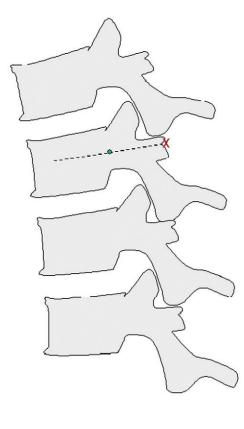

Fig. 31-4 The starting point for thoracic pedicle screws is approximately at the intersection of a line drawn along the superior border of the transverse process and the most lateral aspect of the superior articular process *(red Xs)*. To ensure that the medial pedicle is not breached during insertion of the pedicle finder, the tip of the instrument should not extend beyond the medial pedicle halo on the AP view before it arrives at the posterior vertebral body on the lateral view *(green dots)*.
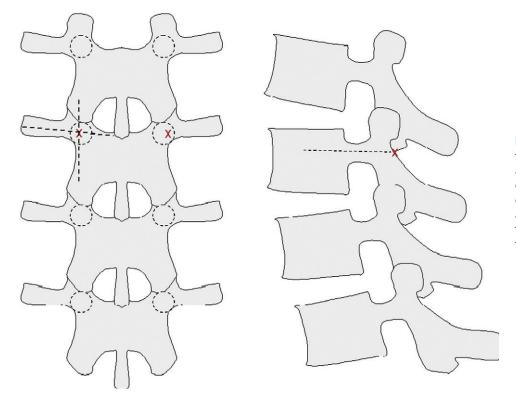
Fig. 31-5 The insertion point for lumbar pedicle screws is at the intersection of a line drawn along the mid-aspect of the transverse process and a line drawn just lateral to the mid-aspect of the facet joint.
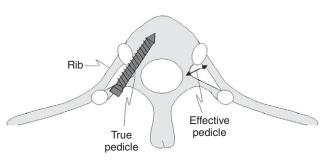
Fig. 31-6 Thoracic pedicle screws can be placed using a lateral extrapedicular insertion technique. This uses the so-called effective pedicle, which considers the rib and the pedicle as one unit. The starting point is more lateral than standard transpedicular screws; the screw must also be more medially angulated.

small and screw insertion technically demanding. There are three hook options that can be used: lamina hooks, pedicle hooks, and transverse process hooks. Lamina hooks can be placed in an upgoing or downgoing direction. Lamina hook placement in the thoracic and lumbar spine requires release of the ligamentum flavum from the bone. Downgoing hooks in the thoracic spine usually require removal of a small portion of the suprajacent overlying lamina to expose the superior border of the lamina to be instrumented. This is a result of the shingled configuration of the laminae in this region.

Pedicle hooks are placed in an upgoing direction only. They are placed through the facet joint and require disrup-

tion of the facet capsule and local stabilizing ligaments. Transverse process hooks are placed in a downgoing fashion. Of the three types of hooks, they offer the least stable point of fixation.

Hooks require compression across opposing "claws" to gain fixation. Until this is achieved, they can be easily dislodged. In our practice, hooks are used as a last-resort fixation method and are placed just before rod insertion.

ROD PLACEMENT AND REDUCTION MANEUVERS

Although screw placement is a critical step, the contour of the rod largely determines the ultimate amount of deformity correction. Precontouring the rod is usually preferred. The rod should be contoured to approximate the normal sagittal curvature of the region instrumented. The thoracolumbar junction is normally straight in the sagittal plane. Thus, a straight rod would approximate normal alignment, which suffices for the majority of thoracic and lumbar fractures as they occur in this region. In the mid thoracic spine, contouring can be more challenging to match the normal or preinjury degree of kyphosis. It is important to avoid over or undercontouring the rod as this can lead to displacement at the injury site. The rod should be straight in the coronal plane to approximate normal alignment.

The surgical challenge is reducing the fractured components. In some cases, gross instability is easily corrected by directly manipulating the fractured segments. Facet joints

can be levered into position or spinous processes can be grasped with towel clamps to effect reduction. Maneuvers should be slow and intentional as to avoid additional neurologic injury.

In some cases, however, reduction requires greater intraoperative forces. These can be applied through pedicle screws using the precontoured rod. In our technique, the rod(s) is(are) fixed to either the proximal or distal aspect of the construct. The free end of the rod can then be reduced into
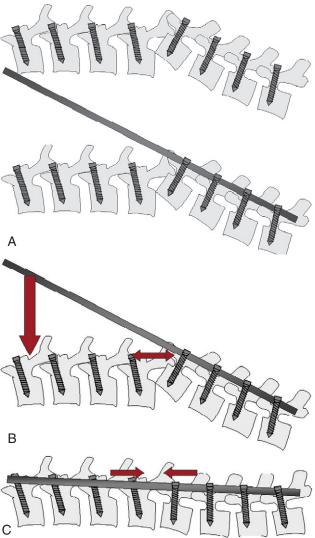

A

B

C

Fig. 31-7 Reduction technique for translational deformities. After placement of three or more levels of pedicle screws above and below the level of injury, a rod is placed and secured to the cranial or caudal region *(A).* The prominent end of the rod is then reduced into the free screw heads *(B).* Distraction between the screws just above and below the injured segment can aid reduction of "locked" dislocations. After the reduction is complete and alignment is confirmed, the construct can be gently compressed *(C).*

the free screw heads (Fig. 31-7, *A-C*). Lock nuts are then used to secure the rod to the screw heads and maintain the reduction. Cross-links can be added in long constructs to maximize torsional stability.

DISTRACTION FOR INDIRECT DECOMPRESSION

A commonly used technique of indirect decompression of retropulsed bone fragments from burst fractures involves distraction. Optimally used within 48 hours from the injury, screws are placed above and below the injury. The rods are then placed and secured tightly into the lower screws. A vise-grip rod holder can then be placed above these screws to act as an anchoring point. A distractor is then used to distract between the vise-grip holder and the top screws to effectively lengthen the construct. This helps restore loss of vertebral body height. It is thought that if the retropulsed fragments are still attached to the posterior annulus or posterior longitudinal ligament (PLL), then ligamentotaxis will pull these fragments back into their native position in the vertebral body.

Posted screws can be useful in this technique, as well. In this maneuver, the posterior tips of the screw posts can be levered toward the injury site to provide distraction and kyphosis correction of the anterior elements (Fig. 31-8, *A*). The rod is then tightened to the screws in the corrected position (Fig. 31-8, *B*). The use of lateral fluoroscopic imaging is useful to monitor intraoperative correction.

FUSION BED PREPARATION

In most cases of posterior surgery for thoracolumbar injuries, the primary initial goal is stabilization. Once this has been performed, attention can be paid toward preparing the fusion bed. Because the long-term goal of surgery is a solid fusion, this is an important step in maximizing the longevity of alignment correction and restoring spinal stability. If not torn by the initial injury, the interspinous process ligaments of the levels to be fused and stabilized are removed with a large rongeur. Importantly, the ligamentous and soft tissue attachments of the adjacent levels not to be fused must be left intact to prevent late deformity from developing. Next, all soft tissues are removed from the posterior aspect of the laminae and facet joints at the levels to be fused. All exposed bone surfaces are then decorticated using a large high-speed burr.

BONE GRAFT HARVEST

Attention is now turned to bone graft harvest. A curvilinear incision is made from the posterior superior iliac spine (PSIS) and extended laterally and superiorly 5 to 6 cm down to the subcutaneous tissue. Care must be taken to avoid posterior cluneal nerves that run approximately 8 cm lateral to the PSIS. Electrocautery is used to dissect down to the deep fascia and the gluteus maximus origin. Using an elevator, a
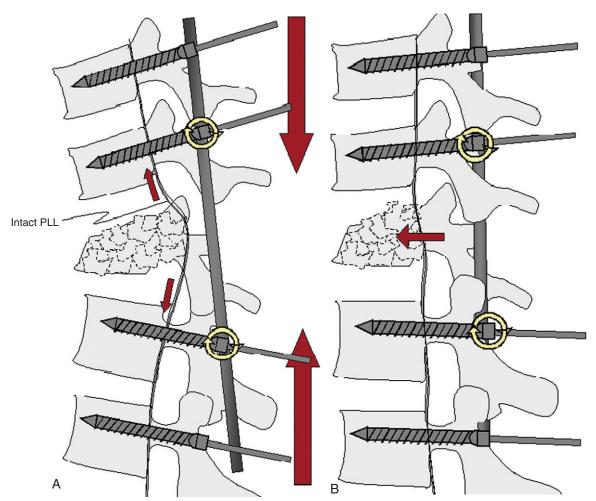
Fig. 31-8 Indirect reduction of burst fractures. Indirect reduction of retropulsed fragments associated with a burst fracture is best performed early (within 24 to 48 hours) and in the presence of an intact PLL. Using posted screws, the post tips can be compressed *(A)* toward the injury site *(large arrows) to* affect distraction of the anterior elements *(small arrows).* The PLL can pull the fragments back into position via ligamentotaxis *(B).*

subperiosteal dissection of the iliac crest outer table is performed. The ligamentous attachments around the sacroiliac joints should be avoided to prevent destabilization. Distally, the greater sciatic notch should not be entered to avoid laceration of the superior gluteal artery.

A corticotomy along the outer table is then made with an oscillating saw. Longitudinal strips of bone are removed with a curved osteotome. Additional cancellous bone is then scooped out with a large curette and placed on the back table. Once hemostasis is obtained, the wound is closed in layers.

FUSION AND CLOSURE

After final irrigation and adequate hemostasis is achieved in the posterior spine wound, the bone graft is packed along the decorticated posterior aspects of the spine. A drain is placed and the wound closed in layers.

OPERATIVE TECHNIQUE: LAMINECTOMY AND REPAIR OF TRAUMATIC DURAL TEARS

In our practice, there are few indications for a laminectomy as a primary means of decompressing a thoracolumbar injury. Rarely is a fragment of fractured lamina or facet joint anteriorly or medially displaced, respectively, into the spinal canal. In such cases, it can be readily removed using a posterior approach via laminectomy. A laminectomy might also be indicated to explore a traumatic dural tear. These often occur with burst fractures that have a concomitant lamina fracture. They may be associated with entrapped nerve roots.

After spinal stabilization has been performed, the lamina in question can be removed. A Kerrison rongeur is used to create a trough on either side of the lamina, just medial to the pars interarticularis (Fig. 31-9). After transecting the
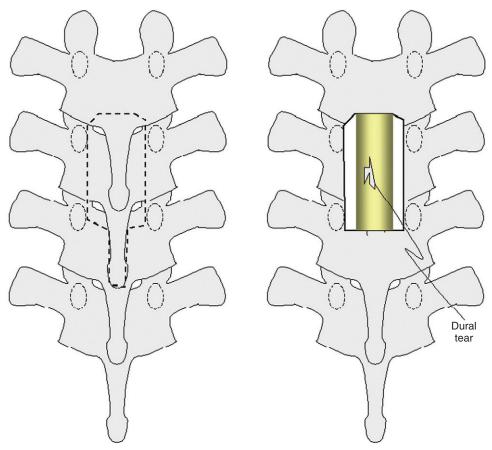
Fig. 31-9 The extent of a laminectomy *(dashed line, left)* should ensure complete visualization of the dural sac. This can often reveal complex traumatic dural tears *(right).*

interspinous process ligaments above and below the lamina (if intact), the lamina can be removed in one piece. In many cases, the lamina is fractured in or more places. Loose pieces can be removed carefully by releasing them from any tethered strands of ligamentum flavum or facet capsule. Dissection in the region of lamina fractures should first be performed with a Penfield 4 elevator to ensure that the neural elements are free.

The endpoint of the laminectomy is an unobstructed view of the posterior dural surface. If a dural tear is noted, the displaced neural matter should be gently reduced into the sac. The tear is then repaired with a running locking 6-0 Prolene suture in a running, locking stitch (Fig. 31-10). As traumatic tears can have complex patterns, water-tight suture closures may not always be possible. Supplementation with artificial dural coverings (e.g., DuraGen) or fibrin glue (e.g., Tisseal) can be helpful.

The exposed dural surface should be protected during placement of bone graft. In our practice, a small wet sponge is placed over the dura until the bone graft is in place. It is removed just prior to closure. A wound drain is avoided if the competency of the dural repair is in question.

OPERATIVE TECHNIQUE: POSTEROLATERAL (TRANSPEDICULAR) DECOMPRESSION

Some surgeons use a more direct technique of reducing retropulsed bone fragments from a posterior approach.[9,44,45] In this maneuver, the pedicle at the injured level as well as the entire hemi-lamina is removed (Fig. 31-11,*A*). The exiting nerve root is protected. Approaching the vertebral body in a posterolateral direction, the retropulsed fragments are pushed forward into the vertebral body using a downgoing curette (Fig. 31-11,*B* and *C*). Adequate midline decompression is difficult to assess in this manner[44] and bleeding can be impressive. In our practice, this technique is rarely used in favor of anterior decompression via corpectomy. The posterolateral technique may have its greatest utility at the T2 to T4 levels in which a direct anterior approach is difficult through a thoracotomy.

Fibrin glue over
artificial dural patch

Dural tear
repaired
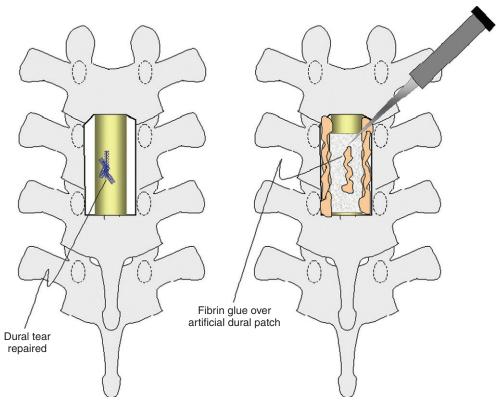
Fig. 31-10 Repair of traumatic dural tears. When possible, dural tears should be primarily repaired. We prefer a running, locking 6-0 Prolene stitch *(left)*. If the integrity of the repair is in question, a synthetic dural patch can be applied followed by fibrin glue application *(right)*.

Pedicle to
be removed

A
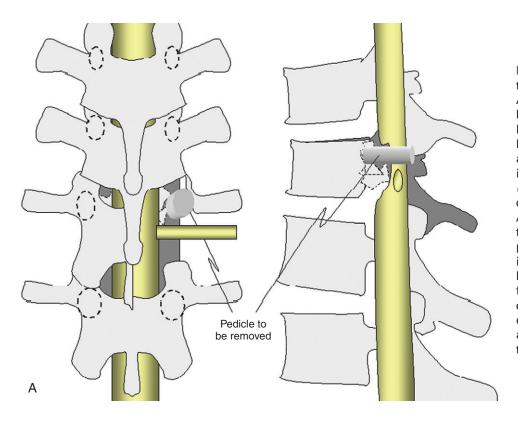
Fig. 31-11 Technique of posterolateral decompression. After pedicle screws have been placed above and below the level of injury, the hemilamina, including the articular processes and pars interarticularis, are removed *(A)*. The pedicle is then excavated and removed *(B)*. A downgoing curette can then be carefully used to push the fragments forward into the fractured vertebral body, *C*. Although this offers better spinal cord decompression than a standard laminectomy, it is associated with substantially greater blood loss.

Downgoing
curette

B

Retropulsed
fragments
reduced

C
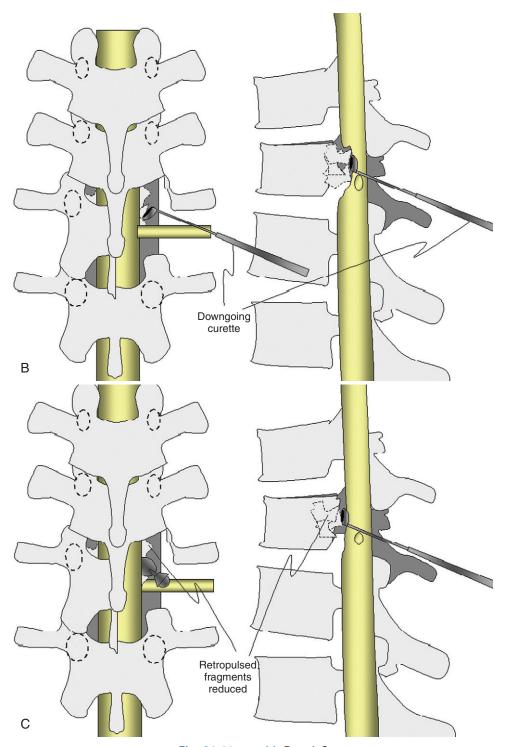
Fig. 31-11, cont'd *B* and *C*

COMPLICATION AVOIDANCE

Surgical techniques and technology for spinal trauma continue to improve, but considerable risks remain present. Neurologic compromise is often an indication for surgical intervention. The entire surgical team must be aware of the potential for additional neurologic insult during the opera-

tive process. This can occur preoperatively, intraoperatively, or postoperatively. Until stability is achieved, strict log roll precautions should be maintained with particular attention during prone positioning. Manipulation of the neural elements or placement of instrumentation can result in mechanical injury. Compression can also occur from a

postoperative epidural hematoma. Some advocate intraoperative monitoring in a patient without neurologic deficits or with an incomplete neurologic injury.

Excessive blood loss may be poorly tolerated in polytraumatized patients with concomitant metabolic or respiratory derangements. Minimizing intraabdominal pressure with positioning, careful attention to hemostasis, and the use of a cell-saver device can be helpful. Transfusion of blood products and replenishment of other factors may be necessary.

Iatrogenic dural tears sometimes occur intraoperatively. They should be repaired if possible. If not, a dural patch may be used, followed by lower lumbar subarachnoid drain placement. Three days of postoperative recumbency can help avoid persistent dural leak.

All operative procedures carry a risk of infection. Rates of postoperative infection in patients who underwent posterior surgery for spinal trauma approximate 10%.[46] Management should include culture-specific parenteral antibiotics, irrigation, and debridement. Hardware should be maintained unless infection persists despite aggressive operative debridement and antibiotics. Adjuncts to debridement may include wound drains, antibiotic-laden cement beads, or vacuum-assisted wound device to enable secondary closure.

The position or integrity of hardware can sometimes be compromised. Metal components can break or subside and screws may pullout. Subsequent loss of the initial correction may then occur. However, such loss of correction does not necessarily negatively affect clinical outcome.[47–49]

In approximately 4% of thoracolumbar trauma cases, a pseudarthrosis may occur.[50] This can be the source of chronic pain and worsening deformity. When pseudarthrosis is suspected, a thorough radiographic analysis should be conducted. An underlying etiology, such as infection, may be present and should also be evaluated with standard clinical findings and laboratory values.

SURGICAL OUTCOMES

COMPRESSION FRACTURES

Long-term pain may be associated with substantial focal kyphotic deformity. This was suggested in a retrospective review by Folman and Gepstein.[51] Eighty-five patients with compression fractures were followed for a minimum of 3 years, revealing chronic low back pain in 69% of cases. Similar results were demonstrated prospectively by Gertzbein.[2]

BURST FRACTURES

Thoracolumbar burst fractures without neurologic deficit managed operatively and nonoperatively have been compared prospectively in two recent studies. In one study,[39] short-segment pedicle screw fixation was compared with hyperextension bracing. Posterior surgery resulted in improved outcome scores at 3 months, but subsequent time periods showed no significant difference. Patients in whom a posterior arch injury was present were excluded. Kyphosis correction and pain scores showed similar patterns. In another study, Wood et al.[40] demonstrated no significant difference when neurologically intact patients without PLC disruption were compared in a randomized, prospective trial. Again, no significant differences were seen in terms of kyphosis, pain, or functional outcome.

Anterior and posterior approaches have also been compared. Readily apparent in using a posterior approach is the avoidance of the increased morbidity associated with anterior exposure. This is particularly salient in patients with concomitant pulmonary or abdominal injuries. Several studies have shown similar functional outcomes between the two approaches, with the posterior approach yielding shorter operative times and decreased blood loss.[52–54] Anterior column support cannot be reconstituted by posterior instrumentation alone, however, which has lead to higher rates of progressive kyphosis and instrumentation failure.[27,55] Some surgeons have advocated the discriminate use of fixation two levels above and below the injured segment, especially in the setting of osteoporosis, severe body comminution, or high stress areas.[41] Alternatively, short-segment instrumentation can be augmented with a combined anterior/posterior approach or transpedicular bone grafting. The latter, however, has yielded disappointing results.[56,57] Although it should be used only as an adjunct to instrumented stabilization, laminectomy has been shown to be an effective decompression technique.[41] The posterolateral method of decompression improves canal clearance compared with standard laminectomy, but it has demonstrated no additional neurologic benefit over posterior instrumentation alone.[58]

FLEXION-DISTRACTION INJURIES

Existing literature suggests that patients with flexion-distraction injuries do well long term in terms of pain resolution. LeGay et al.[59] reported on 17 patients with this injury type, 14 of which were managed nonoperatively. Most were unable to return to preinjury activities, but 80% had only mild or no pain at follow-up. Importantly, patients with significant activity alteration had severe concomitant injuries. Similar pain relief was shown in a report on children by Glassman and associates.[60] No difference was seen between surgical and nonsurgical modalities in their series. Anderson and colleagues'[42] series of 13 patients were managed operatively or nonoperatively based on detailed criteria. Those with more than 15 degrees of kyphosis, a neural deficit, or an entirely ligamentous injury had surgery. Good results were shown in 12 of 13 cases at 1 year. Still other reports have shown good pain resolution in nearly 90% of patients and kyphosis improvement using short-segment stabilization.[8,61]

FRACTURE DISLOCATIONS

The relative infrequency of thoracolumbar fracture dislocations yields a paucity of published outcomes data. Many of the existing reports are based on outdated instrumentation systems that were less dependable than pedicle screw constructs. Diverse mechanisms are also present in these reports, making analysis of any one injury type challenging.[62–64] Spinous process wiring after open reduction may control sagittal displacement, but lateral angulation and displacement are poorly controlled.[65] Harrington rods alone were unable to maintain stability in one series.[7] Contemporary short-segment pedicle screw constructs have yielded conflicting results. Razak and colleagues[66] showed no loss of reduction in a series of 15 patients treated in this manner. Low lumbar injuries failed at a higher rate than thoracolumbar injuries with short-segment fusion.[67]

CONCLUSION

Injuries to the thoracic and lumbar spine are sustained in increasing numbers, primarily as the result of high-energy trauma. Appropriate radiographic identification and understanding of the pathomechanics should guide the spine surgeon's intervention. Although most injuries can be successfully managed with nonoperative treatment, posterior surgery is the most common method of operative treatment. Surgical intervention is generally indicated with neurologic compromise and/or radiographic evidence of instability. Posterior approaches are used primarily for sagittal realignment and stabilization, rather than decompression. However, in some cases laminectomy can be useful for reduction of entrapped nerve roots and repair of dural tears. Likewise, transpedicular decompression is advocated by some and may be a useful alternative to anterior decompression for certain injuries. Pedicle screw constructs are the most popular method of posterior fixation of traumatic thoracolumbar injuries, although hook-based fixation is preferred by some surgeons, particularly in the middle and upper thoracic regions. Regardless of the method of stabilization, reduction maneuvers should be performed with care and precision to avoid iatrogenic nerve root or spinal cord injury.

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Management of Traumatic Thoracic Compression and Burst Fractures

INTRODUCTION

The management of thoracic spine fractures is a much-studied and much-debated topic. Despite the relative infrequency of these fractures, it is critical for the spine surgeon to understand all the treatment options and surgical techniques for managing these often complex injuries. A thorough understanding of the mechanisms of injury, as well as postinjury spinal stability, is critical for proper care of the patient.

Although thoracic and lumbar fractures are often grouped together, thoracic fractures from T2 to T10 are often managed differently than those in the thoracolumbar region. The regional biomechanics of thoracic vertebrae, with stability and immobilization provided by the rib cage, coupled with the presence of the spinal cord, differentiates these fractures from those in the thoracolumbar and lumbar spine.

Although the vast majority of thoracic compression and burst fractures are secondary to osteoporosis, this chapter focuses on traumatic thoracic compression and burst fractures. A complete discussion of osteoporotic compression and burst fractures may be found in Chapter 60.

EPIDEMIOLOGY

Most injuries involving the thoracic and lumbar spine involve males in their second and third decades of life. The approximate incidence is 1 per 20,000. Most are the result of motor vehicle accidents. More than half of the thoracic and lumbar injuries (52%) occur at the thoracolumbar junction (T11 to L1). The lumbar spine (L1 to L5) absorbs the injury 32% of the time.[1] Injuries involving T2 to T10 occur much less often secondary to the rigidity provided by the rib cage

and thoracic musculature. Thoracic injuries represent only 16% of all thoracic and lumbar spine trauma.

Despite the relative infrequency of pure thoracic spine fractures, as compared with the thoracolumbar junction and lumbar spine, thoracic fractures more often result in severe neurologic injury. Between T2 and T10 the spinal cord traverses a region that provides the smallest canal-to-cord ratio in the entire spine, rendering the spinal cord extremely vulnerable to injury. Additionally, the artery of Adamkiewicz courses from superior to inferior adjacent to the thoracic spine before entering the canal, most commonly between the T9 and T12 levels.[2] Injury to this blood supply may result in catastrophic neurologic outcome, although its true significance is still unclear. Because of these factors, combined with the extremely high energy required to fracture these vertebrae, which are supported by the rib cage, there is a higher frequency of complete paraplegic spinal cord injury here than in other regions. The complete to incomplete neurologic deficit ratio is 6 to 1 in severe thoracic spine injuries.[3]

CLASSIFICATION AND SURGICAL INDICATIONS

Appropriate treatment of the patient with a thoracic spine fracture requires an understanding of the regional anatomy, biomechanics, and the stability of the fracture. Although a more complete discussion regarding the mechanisms of injury and pathomechanics of thoracic and lumbar spine fractures is located in Chapter 27, a brief discussion of the pertinent issues with respect to management of thoracic burst and compression fractures follows.

Although many classifications of thoracic and lumbar spine fractures have been proposed, the most important determination is that of stability. White and Panjabi[4] defined spinal stability as "the ability of the spine under physiologic loads to limit patterns of displacement so as to not to damage or irritate the spinal cord and nerve roots and to prevent incapacitating deformity or pain due to structural changes." The goal of treatment of thoracic spine fractures is to maintain the stability of a stable fracture, or to restore stability to an unstable fracture.

Applying White and Panjabi's definition of stability is not always easy or well defined. Multiple other classifications have been proposed. Two commonly cited classifications, those of Denis[5] (Fig. 32-1), and Magerl et al.[6] (AO classification, Fig. 32-2), have been shown to have only moderate reliability and repeatability.[7,8] Furthermore, neither classification adequately addresses soft-tissue injury, neurologic status, or overall stability. In an attempt to create a comprehensive classification that defines injury stability and guides treatment, the Spine Trauma Study Group recently published the Thoracolumbar Injury Severity Score[9] (Fig. 32-3). This system accounts for neurologic status, radiographic appearance of the fracture, and integrity of the posterior ligamentous complex. The authors subsequently proposed a treatment algorithm based on their fracture scoring system (Fig. 32-4). Patients scoring 3 or less are nonoperative candidates. Patients scoring 5 or higher have unstable fractures and are operative candidates. Patients with a score of 4 may be treated either operatively or nonoperatively. Although this classification requires validation in the literature, it may be the most comprehensive classification of the thoracolumbar injuries to date, and it is one of the few classifications that offer a corresponding treatment algorithm.

After assessing the patient and imaging studies, the surgeon must define the fracture as stable or unstable. Most stable fractures may be treated nonsurgically, whereas most unstable fractures are best treated operatively (Table 32-1).

The exception to this rule is a stable fracture with an incomplete neurologic deficit. Although this clinical presentation is much less common in thoracic injuries, as compared with thoracolumbar injuries, anterior decompression of retropulsed burst fragments is often recommended. Additionally, the ability to externally support the level (e.g., T2-T4) must be considered in the management plan. A final consideration is that all nonosteoporotic thoracic through sacral fractures are associated with high forces. The magnitude of the force required to break the thoracic vertebra and ribs may be truly significant, and should be considered when planning management.

NONOPERATIVE TREATMENT

Nonoperative treatment must not be confused with nontreatment. On the contrary, nonoperative treatment should include brace immobilization of the fracture, deep venous thrombosis prophylaxis (initially), aggressive pulmonary toilet, and patient mobilization. Deep vein thrombosis prophylaxis should entail compression stockings and sequential compression devices in all patients without concomitant lower extremity injury. Pharmacologic anticoagulation may be considered if no other patient factors prohibit, and prolonged immobilization may occur because of other injuries. In patients with anticoagulation contraindications, and in patients with significant neurologic compromise, or who are

Compression   Burst

Fig. 32-1 Denis' classification of thoracic and lumbar burst and compression fractures. (Used with permission from Mirza SK, Mirza AJ, Chapman JR, Anderson PA: Classifications of thoracic and lumbar fractures: Rationale and supporting data. J Am Acad Orthop Surg 10:364–377, 2002.)
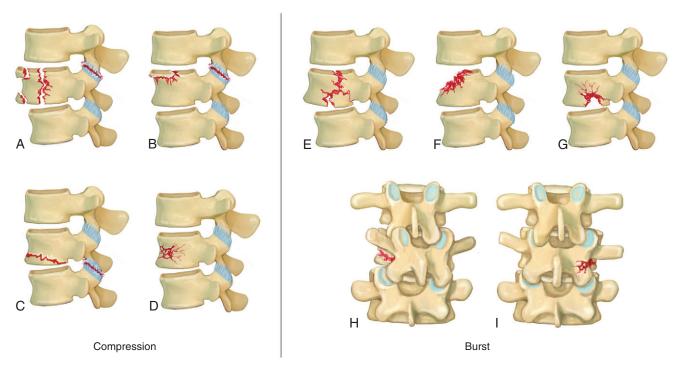
Fig 32-2. Magerl's classification of thoracic and lumbar burst fractures. (Used with permission from Mirza SK, Mirza AJ, Chapman JR, Anderson PA: Classifications of thoracic and lumbar fractures: Rationale and supporting data. J Am Acad Orthop Surg 10:364–377, 2002.)
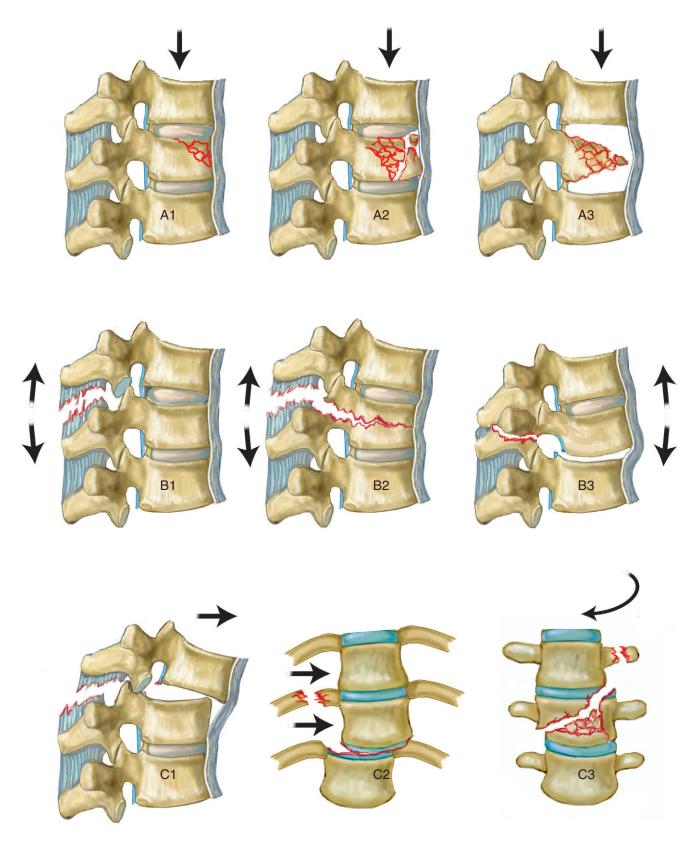
```
                              ┌─ Burst - 1 point
             ┌─ Compression - 1 point ─┤
             │                └─ Lateral angulation > 15
Mechanism of ┤                         degrees - 1 point
  Injury     ├─ Translational/rotation injuries - 3 points
             │
             └─ Distraction injuries - 4 points
```

```
                  ┌─ Intact - 0 point
                  ├─ Root injury - 2 points
Neurologic Status ┤
                  ├─ Spinal cord injury (complete) - 2 points
                  ├─ Spinal cord injury (incomplete) - 3 points
                  └─ Cauda equina injury - 3 points
```

```
Posterior       ┌─ No injury - 0 point
Ligamentous     ├─ Indeterminate injury - 2 points
Complex Injury  └─ Definite injury - 3 points
```
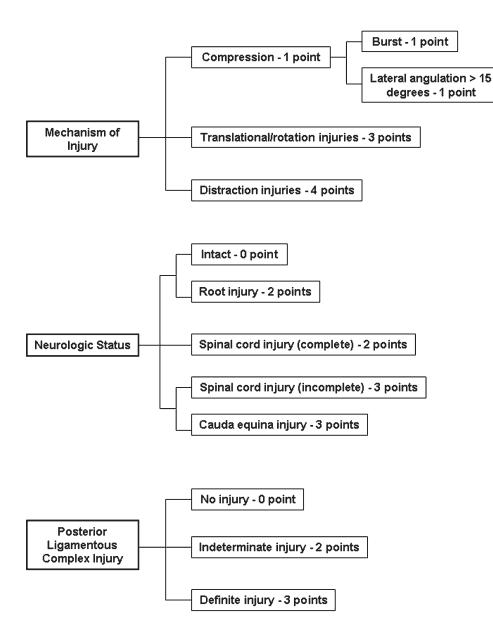
Fig. 32-3 Scoliosis Research Society Injury Severity Score. (Used with permission from Vaccaro AR, Zeiller SC, Hulbert RJ, et al: The Thoracolumbar Injury Severity Score: A proposed treatment algorithm. J Spinal Disord Tech 18:209–215, 2005.)

unable to mobilize quickly, consideration should be made for placement of an inferior vena cava filter in an effort to prevent pulmonary emboli.

Brace immobilization of thoracic fractures may be very effective. A study of 49 patients treated in braces for thoracic compression fractures from T1 to T8 revealed fracture healing in all patients. However, one patient neurologically deteriorated and eight had skin breakdown beneath the braces.[10] When brace management is elected, the brace should be removed regularly for skin checks and neurologic function monitored daily. It is also important to note that thoracolumbarsacral orthoses (TLSO) braces do not immobilize above T5.

The choice of brace immobilization is dependent on the fracture pattern and location. Stable wedge compression fractures from vertebrae T6 to T10 may be treated with either a thoracolumbosacral hyperextension brace, such as a CASH, Jewitt, or a clamshell orthosis. Fractures including and above T5 require the inclusion of cervical immobilization to the TLSO to adequately secure the fracture. For upper thoracic "stable" injuries, a cervicothoracic orthotic with a chest component, such as a Minerva brace, may be adequate. Stable mid- and lower-thoracic burst fractures are best treated with a TLSO. Less stable fractures should have a custom-molded rigid orthotic fabricated.

After brace fitting, patients should wear the brace at all times. They should be mobilized when possible. Postmobilization upright anterior-posterior and lateral radiographs should be obtained to evaluate for further settling of a fracture into unacceptable kyphosis. If the fracture remains stable, mobilization may continue in the brace. The period of bracing usually extends 3 months, or until bone healing has
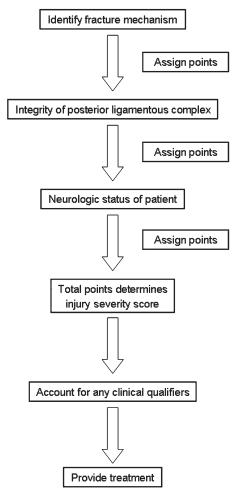
Fig. 32-4 Treatment algorithm for thoracic and compression fractures as proposed by the Scoliosis Research Society. (Used with permission from Vaccaro AR, Zeiller SC, Hulbert RJ, et al: The Thoracolumbar Injury Severity Score: A proposed treatment algorithm. J Spinal Disord Tech 18:209–215, 2005.)

TABLE **32-1**   **Recommended Treatments for Various Types of Thoracic and Lumbar Injuries**

SPINE STRUCTURE	NEUROLOGIC	TREATMENT
Stable	Normal	Nonsurgical
Stable	Complete	Nonsurgical
Stable	Incomplete	Decompression and stabilization: anterior or posterior
Unstable	Complete	Posterior stabilization
Unstable	Incomplete	Decompression and stabilization: anterior, posterior, or both

Adapted from Capen DA: Classification of thoracolumbar fractures and posterior instrumentation for treatment of thoracolumbar fractures. Instruct Course Lect Spine 249–254, 2003.

been radiographically demonstrated. Interval radiographs are recommended to ensure maintenance of coronal and sagittal alignment. At 3-months, postinjury, anteroposterior (AP) and lateral flexion and extension films should be acquired with the patient out of the brace. Lack of motion and the absence of pain at the fracture site indicate a healed fracture and the brace may be discontinued, or a less rigid brace applied, if appropriate. Computed tomography (CT) scans are helpful to assess the fracture because the thoracic vertebrae are often obscured in plain films by the ribs and arms.

TIMING OF SURGERY

If surgical intervention is indicated because of an incomplete neurologic pattern, the surgeon must decide when to perform surgery. This is a matter of debate within the literature. Although animal studies suggest that neurologic recovery may be improved with cord decompression within 6 hours of the initial insult, no human studies prove that early surgery (less than 24 hours from injury time) will benefit neurologic recovery more than delayed decompression.[11,12] Patients with complete paraplegia at presentation secondary to fractures above T10 rarely have any neurologic recovery.[13] When not essential, it is our feeling that middle-of-the-night surgery with an unprepared surgical team may not go as smoothly as midday, scheduled, procedures. The only absolute indication for emergent surgery remains the presence of a documented neurologic deterioration. However, when possible, surgery should be performed within the first 47 to 72 hours postinjury to allow for early mobilization to prevent pulmonary and skin complications.

SURGICAL OPTIONS

Surgical treatment for traumatic thoracic fractures is generally regarded as safe and effective.[14–18] The use of thoracic pedicle screws and development of rigid anterior thoracic spine stabilization instrumentation appear to have improved the surgical treatment of unstable thoracic spine fractures. Although the use of Harrington rods and Luque wiring techniques had reasonable success, studies have shown biomechanical inferiority of these techniques compared with pedicle screw fixation.[19,20] In a review of 70 patients, Sasso et al.[20] reported failure of Luque wiring and Harrington rods to maintain sagittal correction at 12-month follow-up. Korovessis and associates demonstrated the superiority of pedicle screw fixation compared with hooks and rods to maintain alignment, while fusing fewer levels. Thoracic pedicle screws offer the advantage of three-column support from a single approach. Biomechanical studies comparing pedicle fixation with Harrington rods proved pedicle fixation to be more rigid in flexion, extension, lateral bending, and rotation.[21,22] More recent studies suggest that anterior-only fixation may also be adequate for stabilizing even the most unstable fractures.[23]

Despite these new technologies, the goals of surgery remain the same: restoration of alignment, fracture stabilization, restoration of canal anatomy in patients with incomplete neurologic injuries, improvement of neurologic function (if possible), and rapid mobilization. Most authors agree that patients with incomplete neurologic injury, whether or not the fracture is stable, should undergo surgical decompression and/or stabilization of the spinal column.[24,25] Studies suggest that reduction of a traumatically- induced kyphosis by posterior instrumentation may reduce retropulsed fragments in an indirect fashion and offer similar neurologic improvement compared with patients undergoing anterior and posterior surgery.[26–28]

Newer technologies, such as thoracoscopic decompression and instrumentation, may be alternatives to traditional surgical techniques. Currently there is insufficient evidence to claim the superiority of a specific technique. Indications and rationale for each of the surgical options will be discussed independently.

POSTERIOR-ONLY INSTRUMENTATION

Prior to the use of pedicle screws, pedicle hooks and rods in a claw configuration were commonly used to perform rigid posterior fixation. This configuration proved both safe and effective.[29] However, pedicle and laminar hooks extend within the spinal canal, possibly encroaching on the thoracic spinal cord.[30] Additionally, the hook-and-rod construct obscures the laminae and thereby decreases the bone surface available for fusion. Studies comparing hook-and-rod techniques versus pedicle screw-and-rod fixation have demonstrated the superiority of pedicle screw systems with respect to biomechanical rigidity, fracture reduction, maintenance of reduction, and reduced complications from instrumentation pullout.[19,21,22]

Roy-Cammille et al.[16] were the first to describe the use of pedicle screws with plate instrumentation for the treatment of thoracic and lumbar spine fractures. Since that time, the safety and efficacy of pedicle screws and posterior rod instrumentation have been established for the treatment of thoracic and thoracolumbar fractures.[15–18] The advantage of pedicle screw-and-rod systems is that they provide three-column support via a posterior-only approach. Although most of the studies evaluate thoracolumbar fractures, a recent study followed 32 patients with 79 vertebral fractures from T2 to L1.[15] The authors had no cases of loss of reduction or painful hardware removal. They found that posterior-only instrumentation was sufficient. Their patients maintained an average improvement in kyphosis of 62% compared with their preoperative status. These same patients exhibited a postoperative decrease in canal compromise of 8% of overall canal diameter (35.4% canal compromise to 27.4% compromise with a $p < .7$) by posterior-only reduction. This study shows that posterior-only reduction and instrumentation may result in indirect reduction of retropulsed burst fragments. Crutcher et al.[31] evaluated 42 patients with thoracolumbar burst fractures preoperatively and postoperatively. They found approximately 50% reduction in canal compromise with indirect reduction techniques. Similarly, Sjostrom et al.[26] evaluated the amount of indirect decompression they were able to obtain by posterior-only fixation in 67 consecutive patients with burst fractures from T12 to L2. They also found approximately 50% reduction in canal compromise over their preop CT. It should be noted, however, that the latter two studies evaluated fractures at the thoracolumbar junction and not in the thoracic spine. In summary, multiple studies support the use of posterior-only pedicle screw-and-rod instrumentation for the treatment of traumatic thoracic compression and burst fractures.

ANTERIOR-ONLY INSTRUMENTATION

Disadvantages of the posterior-only approach include inability to completely reduce the retropulsed fragments of a burst fracture, and perhaps less rigid anterior column support that may result in late kyphosis. This led Dunn and Kaneda to develop rigid anterior fixation devices for thoracic and lumbar burst fractures.[32–34] Subsequently, a biomechanical study performed in a calf model comparing anterior and posterior fixation techniques in the thoracolumbar spine revealed that short-segment, posterior-only fixation was less rigid in flexion and extension than anterior-only fixation with the Kaneda system with a strut graft.[35] Multiple other studies have confirmed the efficacy of anterior fixation with strut grafting in thoracic and thoracolumbar burst fractures.[23,34,36–38] Sasso et al.[23] published their results on 40 patients treated with anterior decompression, strut grafting, and instrumentation. They found only one construct failure, which was due to a technical error. Average reduction of the kyphosis improved from 23 degrees preoperatively to 7 degrees postoperatively. Just as important, the average increase in kyphosis at follow-up was only 2 degrees, illustrating the stable nature of this construct. They had no cases of neurologic worsening. Ninety-one percent of patients with incomplete neurologic deficits improved by at least one Frankel grade.

A relatively new twist on anterior-only procedures, in which a minimally invasive thoracoscopic approach is used, has been advocated by Dickman and others.[39–42] The reported advantage of a thoracoscopic approach, although technically challenging and initially more time consuming, is a decreased complication rate over the traditional thoracotomy.[43] Until recently, one disadvantage was that instrumentation could be performed only with screw-plate systems that were less biomechanically sound than traditional dual-rod systems. However, Horn and colleagues[42] recently published two cases in which they implanted dual-rod instrumentation via the thoracoscopic approach. Despite their early success with these initial patients, future studies are required to assess the long-term efficacy of this procedure.

COMBINED ANTERIOR AND POSTERIOR SURGERY

Despite the many studies proving the long-term efficacy of anterior-only and posterior-only procedures for the treatment of thoracic spine fractures, occasionally anterior and posterior fusions are required to obtain appropriate stability. Poor bone quality, a high degree of instability, and/or location of neural impingement may necessitate combined anterior and posterior procedures with fusion.[44] Shiba and associates[45] reported a 98% fusion rate with combined anterior and posterior surgery for thoracolumbar burst fractures and their patients improved by an average of one Frankel grade.[20] Combined surgery may also be indicated when a retropulsed fragment necessitates decompression, but anterior fusion is contraindicated or tenuous because of osteoporosis or concomitant fractures.[44]

Posterior-only surgery may also fail to maintain sagittal correction in unstable fracture patterns. One study evaluated three different posterior systems, including a pedicle screw and plate construct, hooks and Harrington rods, and sublaminar wires with Luque rods. All failed to maintain sagittal balance at 12-month follow-up, indicating a possible benefit of anterior column support.[20,44]

If combined surgery is indicated, the sequence of procedures is determined by patient-specific factors and fracture characteristics.[44] In general, for thoracic burst fractures without a dislocation component, anterior decompression and stabilization is performed first, followed by posterior instrumentation and fusion. Patients with a concomitant dislocation often require posterior reduction, fusion, and instrumentation first, followed by anterior decompression, strut grafting, and stabilization. In severe injuries and in patients with poor bone quality, a combined anterior and posterior approach can be an appropriate and efficacious approach to treatment.

KYPHOPLASTY FOR TRAUMATIC FRACTURES

Kyphoplasty is currently a well-established treatment for painful osteoporotic vertebral compression fractures.[46–48] The advantages of kyphoplasty over vertebroplasty include improvement in vertebral body height and a decreased chance of cement leakage outside the vertebral body.[49] However, the use of vertebroplasty or kyphoplasty in traumatic compression and burst fractures are not as well-studied. Chen and Lee[50] recently published a report on six patients with stable traumatic burst fractures treated with vertebroplasty. Prior to vertebroplasty, each patient had persistent pain after at least 3.5 months treatment in a TLSO. Pain scores were improved in all patients postoperatively, but cement leakage occurred in four of the six patients. A recent study evaluated the efficacy of kyphoplasty for the treatment of traumatic thoracolumbar fractures.[51] Kyphoplasty was performed for 10 compression fractures

(Magerl A1,) 2 split fractures (Magerl A2), and 1 burst fracture (Magerl A3). Injected material was either polymethylmethacrylate (PMMA) (in patients >50, seven patients), or tricalcium phosphate (in patients <50, five patients). All patients experienced nearly immediate pain relief, even without the use of a postoperative orthosis. At 4-month follow-up, stabilization was maintained. Kyphoplasty alone may have a role in the treatment of traumatic compression and burst fractures, but long term, conclusive studies are lacking.

A current trend in kyphoplasty is to combine it with open techniques. A recent study demonstrated that patients undergoing posterior indirect reduction (posterior pedicle screw-and-rod placement) combined with kyphoplasty and PMMA injection for vertebral body burst fractures had excellent maintenance of vertebral body height, kyphosis reduction, and endplate reduction.[52] The kyphoplasty technique may provide improved anterior column support during fracture healing and while the posterior fusion is maturing.

Although these studies are promising, randomized, controlled clinical trials comparing kyphoplasty techniques to more traditional methods for the treatment of traumatic thoracic compression and burst fractures have yet to be performed.

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C H A P T E R

33

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Fracture Dislocations of the Thoracic Spine

INTRODUCTION

Fractures of the thoracic spine from T2 to T10 need to be differentiated from those more cephalad and caudad. Fractures that occur more cephalad represent cervicothoracic injuries and behave more like cervical fractures. Fractures that occur distal to T10 are classified as thoracolumbar fractures. The spine between T2 and T10 is buttressed by the sternum and the thoracic cage and has different biomechanical characteristics. This chapter focuses on the fracture dislocations that occur within the region of the vertebral column between T2 and T10.

In this area, unique anatomic considerations confer increased stability to the thoracic spine. First, the configuration of the laminae, which are broad and overlap, the facet joints, which are oriented in the coronal plane and limit anterior translation, and smaller intervertebral disks all allow less motion in the thoracic spine.[1] Second, the vertebral column gains additional support from the sternum and the thoracic cage. Berg[2] described the sternum as the fourth column of the thoracic spine, which expanded the three column theory presented by Denis.[3] The presence of the rib cage increases the mechanical strength of the thoracic spine fourfold.[4] Because of this additional support, significant energy is necessary to cause bony and/or ligamentous injury in this region of the spine. Consequently, a high degree of neurologic injury occurs. Hanley and Eskay[5] found that fractures in this region constitute 16.4% of all thoracic, thoracolumbar, and lumbar spine fractures treated at their institution during a 10-year period. They also noted that more than 50% of these patients had neurologic injury. The rate of neurologic injury is even higher in association with fracture dislocations.[6] Bohlman et al.[7] reported a series of 218 patients with upper thoracic trauma and neurologic injury. They found that 84% of the patients had complete and permanent neurologic injury.

Another distinguishing feature of thoracic spine fracture dislocations is the potential for concomitant solid organ injury. Although literature documenting the rates of these associated injuries is lacking, vigilance is required in patient workup. At our institution, particular attention is paid to potential kidney, splenic, and liver lacerations; aortic and vena cava injuries; and lung contusions. These associated injuries often dictate when and how surgery is performed.

CLASSIFICATION

Thoracic fracture-dislocations are best described by the classification system presented by Magerl et al.,[8] who proposed three basic injury patterns: compression (type A), distraction (type B), and multidirectional with translation (type C). The basic patterns are then subdivided based on severity. This classification was later adopted by the AO/ASIF and modified into its current form.[9] Thoracic spine fracture dislocations are type C injuries.

Hanley and Eskay[5] describe a subset of injuries termed *burst-dislocations.* Burst-dislocations comprise fractures that display components of both a burst fracture and a dislocation (Fig. 33-1). In their series of 58 thoracic spine fractures, fracture-dislocations and burst-dislocations comprised 49.1% and 5.2%, respectively.

DIAGNOSTIC TESTS AND RADIOGRAPHS

Radiographic examination of the thoracic spine can be challenging and thus requires special attention. Long exposure of 3 to 6 seconds at 50 mA and 70 kVp, combined with a halt in respiration, can increase the quality of the lateral thoracic radiograph.[1] A swimmer's view, usually used to assess the lower cervical spine and cervicothoracic junction, is helpful in showing the upper thoracic spine.[1] Examination of radiographs must include assessment of both the bone and soft tissue elements. Alignment of the thoracic spine in the anteroposterior and lateral plains must be scrutinized for sudden changes in alignment (Figs. 33-2, *A* and *B*). Additionally, any abnormal kyphosis, pleural fluid, rib fractures, sternal fractures, costovertebral dislocations, widening of the interpedicular distance, or paraspinal swelling should be noted.[1] Dennis and Rogers[10] reported that 69% of patients with
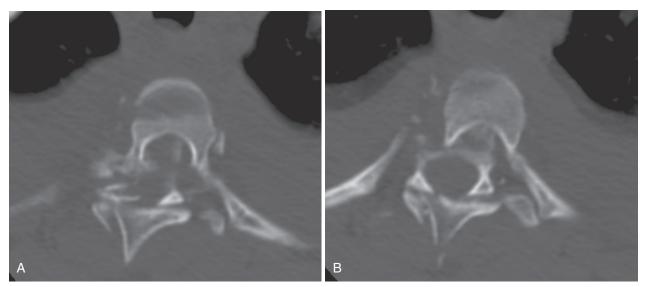
Fig. 33-1 Axial view computed tomographic scans show a burst-dislocation. On these two consecutive axial view scans, note the comminution of the inferior vertebral body associated with anterior translation of the superior body.

thoracic fracture and/or dislocation have mediastinal widening. They noted that a widened mediastinum associated with fracture of the upper thoracic spine likely results from paraspinal muscle swelling. However, a high index of suspicion must be used to rule out aortic injury.

When examining the radiographs, associated injuries of the sternum also must be noted. Thirteen percent of patients with sternal fracture have spinal fracture, and more than half of these injuries are located in the thoracic spine. If the sternal fracture is displaced, the incidence of spinal injury increases to 35.3%.[11] The pattern of the sternal injury might indicate whether it is the result of indirect or direct trauma. Indirect sternal injuries usually manifest with the superior portion of the sternum displaced posteriorly because of the effect of the ribs. Conversely, direct sternal injuries show the inferior sternal fragment displaced posteriorly.[1]

Additional images can be obtained with computed tomography (CT) and magnetic resonance imaging (MRI). CT can be especially helpful in the upper thoracic spine, where the shoulders can interfere with adequate radiographs. In the setting of severe trauma, CT is necessary for any area of concern revealed by plain radiography. Thin sections obtained at 2 mm or less should be augmented with coronal and sagittal reconstructions. These scans help evaluate the bony alignment and help with preoperative planning and understanding the "personality" of the fracture (see Fig. 33-2).

Often, MRI is not needed for fracture dislocations but might be useful in identifying injury to the spinal cord itself and can help define the possibility of neurologic involvement and recovery. Involvement of the posterior column is nearly universal in fracture dislocations.[3] Bright signal on

T2-weighted images represents myelomalacia and cord edema. However, dark signal within the cord on T2-weighted images represents hematoma. Cord edema usually represents a better prognosis than does hemorrhage. Schaefer et al.[12] and Selden et al.[13] noted that patients with incomplete spinal cord injury and evidence of edema but not spinal hematoma improved to a greater degree than did patients with spinal cord hematoma. MRI is most useful for patients who are neurologically intact or incomplete to evaluate discoligamentous injury and areas of continued compression. Neurologically intact patients with large herniated disks might benefit from an anterior approach to decompress the disk and protect the neural elements similar to the cervical spine.[14] Care must be taken when transporting these potentially unstable patients to the MRI suite. Unlike plain radiography or CT, MRI places the patient in isolation, away from immediate access to health care providers. Image acquisition with MRI is substantially longer than with CT or radiography modalities. Therefore, MRI often is not indicated for unstable polytrauma patients.

CLINICAL MANAGEMENT

Because of the anatomic construction of the thoracic spine, patients who sustain dislocations in this area have suffered high-energy trauma. Associated life-threatening injuries need to be addressed concomitantly with the spinal injury. Although the patient is awaiting definitive treatment, one must adhere to spine precautions and be proactive in the prevention of complications such as decubitus ulcers and deep vein thrombosis. We recommend the use of a roto-rest bed, which allows for gentle, continuous turning of the patient and pressure relief while
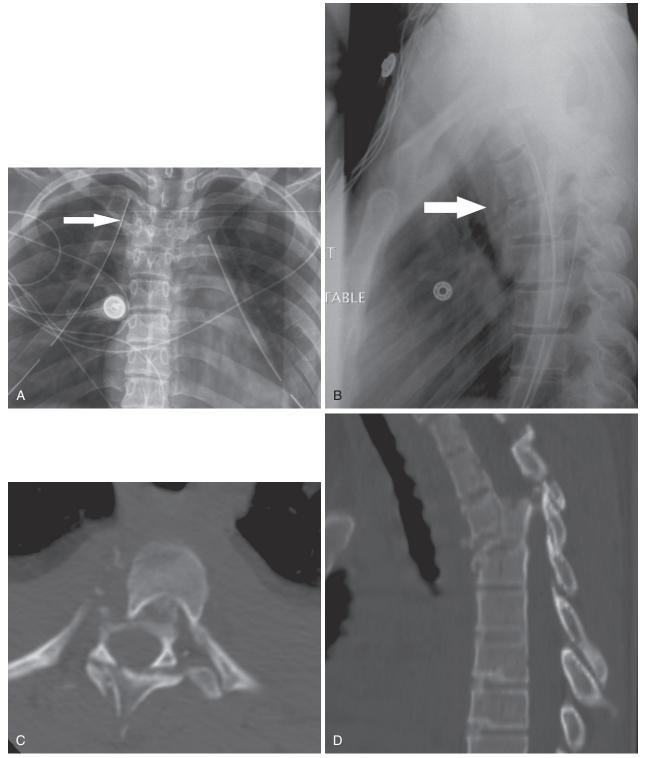
Fig. 33-2 Images of an 18-year-old female patient who was involved in a motor vehicle accident. *A,* Note the break in spinous process alignment in the upper thoracic spine *(arrow),* indicating an injury at that level. *B,* Lateral view radiograph of the upper thoracic spine is difficult to interpret. A subtle line shows the collapse of the T4 vertebral body *(arrow). C,* Axial view scan show the burst component of the injury. *D,* Sagittal CT reconstructions allow for better appreciation of the degree of bony destruction and malalignment of the thoracic spine.

minimizing patient movement. However, gentle log rolling every 2 hours is an alternative if a roto-rest bed is not available in a timely manner. Compression stockings and sequential compression boots should be considered for deep vein thrombosis prophylaxis. At our institution, chemical prophylaxis is held until the surgical treatment plan is determined. Patients should receive an indwelling urinary catheter to prevent bladder distension caused by spinal cord dysfunction and the need for patient manipulation during micturition. A nasogastric tube is placed for gastric decompression to decrease the risk of aspiration. Additionally, gastric prophylaxis against ulcers, especially with the use of trauma dose steroids, should be administered. Finally, aggressive bowel programs are necessary to prevent constipation and autonomic dysreflexia.

SURGICAL INDICATIONS

The decision regarding surgical versus nonsurgical management of fracture dislocations is individualized considering the extensive nature of concurrent injuries in this patient population. The goals of surgical intervention are as follows: (1) spinal cord decompression, if needed; (2) maintenance or re-establishment of spinal alignment; (3) achievement of spinal stability; and (4) early mobilization, early rehabilitation, and decreased hospitalization to diminish the risk of secondary complications. Nonoperative treatment of these injuries often requires long periods of bed rest and results in significant morbidity. Unlike bony burst or compression fractures, thoracic dislocations and fracture dislocations result in significant ligamentous injury and poor healing with nonoperative treatment. Therefore, nonoperative management should be considered for only those rare patients whose associated injuries prohibit surgical management.

OPERATIVE PROCEDURES AND TECHNIQUES

Operative intervention can be undertaken in multiple manners. Posterior, anterior, and combined anterior-posterior approaches have been described for thoracic trauma. Posterior approaches are most appropriate for neurologically complete patients because the goal of surgery is spine stabilization and not decompression. An anterior approach is indicated for the neurologically incomplete or intact patient with persistent spinal cord compression and/or spinal malalignment. It also is used when insufficient anterior column support is present. In most cases, anterior surgery is supplemented with posterior fixation. Each patient must be individually evaluated for the proper approach. Regardless of the approach, preoperative planning is essential for both fracture fixation and patient positioning. The patient should be placed on a radiolucent table to facilitate intraoperative imaging. The use of image guidance is helpful for pedicle screw insertion, particularly at the upper thoracic levels.

Consideration should be given to intraoperative neurophysiologic monitoring with both sensory and motor-evoked potentials in the neurologically intact or incomplete patient. Electrophysiologic monitoring has received much attention for elective spine procedures in recent years. However, no literature specifically related to its use in thoracic spine trauma is available. Padberg and Bridwell[15] reviewed their recommendations for monitoring based on the operative spinal levels. For posterior thoracic surgery from T2 to T12, they recommend the use of ulnar and tibial somatosensory-evoked potentials and motor tract monitoring. Similar monitoring should be considered for anterior surgery at these levels, although no specific recommendations have been established. Raynor et al.[16] examined the potential of triggered electromyographic thresholds in the rectus abdominus muscles to monitor thoracic pedicle screw placement from T6 to T12. They found reliable prediction of adequate screw placement when using a less than 6.0-mA threshold and when no single screw showed a threshold that was less than 60% to 65% of the thresholds of all the other screws. Caution should be used when applying the data to the trauma population because the population studied was predominantly a spinal deformity population.[16] At our institution, somatosensory-evoked potentials, neurogenic motor potentials, and anal sphincter monitoring are used in all cases except those with complete spinal cord injury. Neurophysiologic monitoring is mainly intended to assess spinal cord function during the reduction maneuver. If a loss or change in signal is noted during reduction, the maneuver is halted and additional decompression is performed as necessary to facilitate safe realignment of the spine.

In all cases, preoperative planning is required to ensure that all appropriate equipment is available in the operating room. The surgeon should verify that small-diameter, low-profile implants are available for insertion into the upper thoracic pedicles. Additional fixation devices, such as hooks and wires, should be available in case pedicle screws cannot be placed safely. Furthermore, dural tears might be more common with fracture dislocations. Provisional plans for repair, including tensor fascia lata patch grafting, should be in place.

POSTERIOR APPROACH

The aim of the posterior approach is to reestablish the posterior tension band. This approach avoids violation in the pleural cavity with either thoracotomy or thoracoscopy. Additionally, the approach is familiar for the majority of spine surgeons. Posterior-only procedures are best for complete spinal cord injuries that require stabilization (Fig. 33-3). The goal of surgery for the patient with complete neurologic injury is to expedite mobilization and rehabilitation during the early postoperative period.

In 1985, Bohlman[17] reported the poor results of patients with upper thoracic spinal injuries and incomplete neurologic lesions treated with posterior-only procedures. Of 17 patients with incomplete spinal cord injury who underwent posterior-only procedures with or without fusion, 8 had

either no change or worsening of the results of neurologic examination after posterior surgery. Laminectomy alone is contraindicated in patients with fracture dislocation or dislocation because of the risk of postlaminectomy kyphosis. Additionally, although not explicitly mentioned in the report presented by Bohlman, the majority of spinal cord compression is anterior in such cases and decompression from a posterior approach can be challenging.

The ability to treat thoracic fracture dislocations with a posterior approach has improved with segmental fixation that uses pedicle screw constructs. This pedicle screw technique is an extension of what has been used with success in the lumbar spine. The technique provides increased reduction strength and deformity correction.[18,19] Additionally, these screws do not enter the spinal canal, unlike sublaminar wires and hooks. Polly et al.[20] examined the amount of canal intrusion with pedicle screws or hooks. They found that screws need to violate the medial wall of the pedicle by more than 3 mm before the canal compromise is similar to that of the largest set of hooks. Similarly, more than 1.5 mm of medial violation is necessary to equal the space occupied by the smallest pediatric hooks.[20] Finally, pedicle screws allow posterior fixation of the spine in the absence of intact posterior elements.[19]

The placement of thoracic pedicle screws can be technically demanding and requires particular attention to anatomic detail. The pedicles in the thoracic spine are much smaller than those in the lumbar spine. Yue et al.[19] evaluated 32 patients with thoracic and lumbar spinal injuries that resulted from both low- and high-energy trauma. At an average follow-up of 22 months, they found that the average sagittal index correction, defined as the deviation of the sagittal alignment in a motion segment from the normal value, was 62%. The Gardner segmental kyphotic deformity, defined as the kyphosis across the fractured vertebra and the level above and below, improved 33.3%. They also noted fusion at an average of 4.8 months. No implant failures or intraoperative complications occurred as a result of implant placement. Carbone et al.[21] examined 22 of 41 patients who were treated with pedicle screw fixation with the use of CT. Although they found that 12.7% of the screws had penetrated either the medial or lateral pedicle, no cases of iatrogenic neurologic injury were included. These findings show that pedicle screws are a safe means of thoracic fixation and are likely more efficacious for the treatment of unstable injuries.

Although most complete neurologic injuries do not require decompression, both the costotransversectomy and the lateral extracavitary approaches allow better access to the anterior column than does a direct posterior approach. Both can be used to decompress the spine anteriorly. The lateral extracavitary approach can provide adequate visualization of the anterior column, not only to decompress the anterior spine but also to place anterior strut grafts.

Several technical aspects of the surgery can facilitate anterior column access. First, one or more thoracic nerve roots might be ligated to aid anterior column access. Typically, paresthesias and pain are not significant complaints postoperatively. Second, removal of the rib head allows a far lateral approach to the vertebral body. Usually 1 cm of the rib head is removed to gain successful visualization of the anterior column. Finally, reconstruction of the anterior column requires a solid bone graft to bridge across the resected segment. Expandable cages facilitate placement of anterior column support through the posterolateral portal of either a costotransversectomy or lateral extracavitary approach. These cages can be placed through a smaller working window and can then be expanded to fill the entire anterior column defect.

ANTERIOR APPROACH

The anterior column of the spine can be accessed by using either an open or thoracoscopic approach. These approaches allow direct anterior decompression of the thoracic spine and strut graft placement and instrumentation. Although helpful for burst fractures, thoracoscopic approaches for fracture dislocations are not well described. Anterior approaches are best suited for neurologically intact or incomplete patients with evidence of anterior spinal cord compression. Although the anterior approach allows for optimal spinal decompression, instrumentation also is possible. In the lower thoracic spine, the approach is typically from the left side because the aorta is less prone to injury relative to the vena cava. However, in the upper thoracic spine, a right-sided approach generally is preferred to avoid the heart and aorta, which encroach on the surgical field. Double-lumen ventilation is advisable for one lung to be deflated during the procedure. These double-lumen tubes are large and need to be exchanged postoperatively. Therefore, one must be cognizant of excessive airway swelling secondary to fluid overload during surgery that might preclude this exchange. One must also be aware of the segmental vessels that cross each vertebral body along with its corresponding nutrient foramen. Both can lead to substantial bleeding if not ligated and sealed. During the approach, one rib can be removed, which can be saved for bone graft augmentation.

Kostuik[22] reported the results of anteriorly treated thoracic and lumbar fractures. He found that 40 of 42 patients who have fusion and were most neurologically incomplete improved their function at least one Frankel grade. The average improvement in the patients treated early (within 10 days of injury) was 1.6 grades. Twelve of 13 patients with bowel or bladder dysfunction recovered useful function. Similar to other reports, none of the complete spinal cord injuries improved. These findings emphasize the poor prognosis for these patients. Sasso et al.[23] reviewed 40 patients who underwent anterior-only fixation for three-column spinal injuries and found good kyphotic correction. They concluded that three-column injuries can be addressed with anterior-only fixation. However, these injuries did not include rotational or translational injuries, which possess greater instability. Caution is warranted against applying these results to
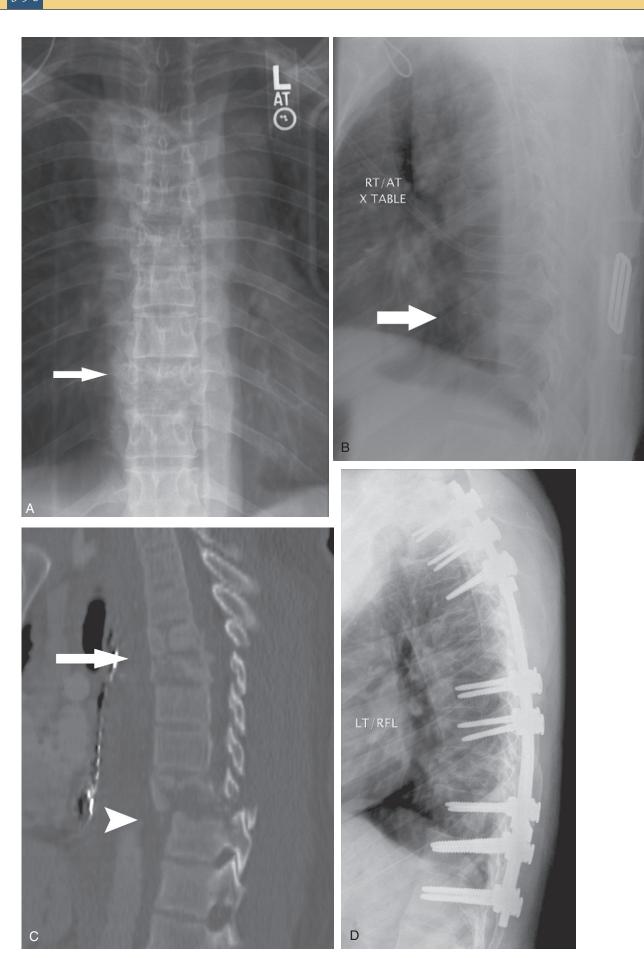
Fig. 33-3 Imaging studies of a 28-year-old male patient who fell 25 feet. He presented to the trauma department with complete neurologic injury. Workup revealed multiple spinal fractures at T5-T6 and a fracture-dislocation at T9-T10. Radiographs show widening of the interpedicular distance *(A, arrow)* and displacement in the midthoracic spine *(B, arrow). C,* Sagittal CT reconstruction is needed to fully show the fractures at T5-T6 *(arrow)* and the burst-dislocation at T9-T10 *(arrowhead). D,* Postoperative lateral view radiographs show realignment of the spine and fixation across both the dislocation area and the upper burst fracture.

fracture-dislocations. Although no literature directly addresses this subject, a general consensus is to augment anterior decompression and fusion with posterior instrumentation for patients with thoracic dislocations and fracture dislocations. In highly unstable injuries, posterior fixation can be performed first to stabilize the spine before extensive anterior column resection.

CONCLUSION

Because of the unique anatomic characteristics of the thoracic spine and the additional support supplied by the rib cage and sternum, thoracic fracture dislocations involve high-energy mechanisms that often result in neurologic injury. Initial management includes appropriate Advanced Trauma Life Support before and immediately after arrival at the hospital. Along with proper resuscitation, suspicion for concomitant solid organ or vessel injury is warranted. Thoracic dislocations and fracture dislocations involve all three columns and are highly unstable. Therefore, most are treated operatively. The correct approach must be tailored to the individual case; posterior procedures or combined anterior-posterior procedures are most commonly used. The goal is to provide a stable spine and to prevent further neurologic injury or late deformity, thus allowing the patient early mobilization and an expeditious recovery.

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Thoracolumbar Compression and Burst Fractures

INTRODUCTION

Traumatic fractures of the spine occur most commonly at the thoracolumbar junction, largely because of the transition from the relatively stable thoracic spine to the more mobile lumbar spine. Common fracture types include compression and burst fractures, flexion-distraction injuries, and fracture dislocations. Compression fractures are the most common type, but burst fractures are more severe injuries and their treatment remains somewhat controversial. This chapter focuses on these two types of thoracolumbar injuries, with a special emphasis on treatment of burst fractures for which a clear consensus does not exist.

MECHANISM OF INJURY

Understanding the mechanism of thoracolumbar fractures is important. It allows one to gain insight into the type of fracture present, associated soft tissue injuries, and the stability of the injury. This then helps in making appropriate treatment decisions.

The different forces that cause spinal injuries include axial loading, flexion, extension, lateral compression, flexion distraction, flexion rotation, and shear forces. This section concentrates on the forces that result in compression and burst fractures. Burst fractures typically are the result of axial loading with or without flexion forces, although several different forces can be involved in these injuries. The anterior and middle columns fail under compression. Compression fractures are due to collapse of the anterior column, usually with no damage to the middle or posterior column. The lack of middle-column injury differentiates compression fractures from the other fracture types. In severe cases, there may be a partial tension failure of the posterior column.

Axial loading can result in a varying degree of injury severity. Axial loading in the thoracic spine more often results in anterior flexion forces on the vertebral bodies because of the normal kyphosis of the thoracic spine. In the thoracolumbar spine, where the spine is straighter, an axial load results in a more uniform compressive force. As this force increases, fractures first occur in the vertebral endplates, followed by vertebral body compression and wedge fractures, and finally burst fractures occur.[1,2] With continued axial loading, centripetal forces result in retropulsion of bony fragments into the spinal canal. Heggeness and Doherty[3] documented an array of trabeculae that originates from the medial corner of the base of the pedicles and extends in a radial array throughout the vertebral body. The cortex of the vertebral canal thins abruptly near the base of the pedicle, which corresponds with the origin of this trabecular array, creating a stress concentration at this site. This anatomy may explain why the retropulsed bone fragments in burst fractures are often trapezoidal in shape.

Further axial loading can cause fractures at the junction of the vertebral body and pedicle, which results in widening of the interpedicular distance. With severe axial loading or with added flexion, posterior element disruption can occur as a portion of the load is absorbed by the facets. This force though the facets can be transmitted to the lamina and pedicler which results in fractures of these structures and further instability of the injury.

CLASSIFICATION

Several classification schemes have been developed for fractures of the thoracolumbar spine, but there is no universally accepted classification system. One of the first systems was proposed by Holdsworth,[4] which characterized spinal fractures based on a two-column anterior and posterior model of the spine. The two most widely used systems today are the subsequent Denis System and the Comprehensive Classification System.

The classification proposed by Denis[5,6] in 1983 is based on a three-column concept of the spine. It introduced the concept of the middle column, formed by the posterior half of the vertebral body, the posterior longitudinal ligament,

and posterior annulus fibrosis. He postulated that this column was vital as its mode of failure correlates with both the type of fracture and neurologic injury. The major injuries were grouped into four categories: compression fractures (48%), burst fractures (14.3%), seatbelt injuries (flexion distraction) (4.6%), and fracture dislocations (16.3%). Because this chapter focuses on compression and burst injuries, only these types are discussed in detail here.

Compression injuries are fractures of the anterior column with an intact middle column caused by axial loading. The posterior column may be disrupted if there are also concomitant anterior or lateral flexion forces, causing compressive loading in the anterior column with tensile strain across the middle and posterior columns. Compression fractures are further subdivided into anterior and lateral.

Burst fractures are caused by an axial load that results in compressive failure of the anterior and middle columns. This can lead to widening of the pedicles and retropulsion of bone into the spinal canal. Denis et al.[7] further divided thoracolumbar burst fractures into five subtypes in 1984. The main deforming force in all types is an axial load. Type A fractures involve fracture of both the superior and inferior endplates and are usually seen in the lower lumbar spine. Type B fractures involve only the superior endplate and are usually seen at the thoracolumbar junction. These are the most common. Type C fractures involve only the inferior endplate and are rare. Type D fractures involve a burst fracture of the middle column with rotational misalignment resulting from axial loading with a rotational force. Type E fractures are the result of an axial load with lateral flexion and are associated with an asymmetric loss of vertebral height. These differ from lateral compression fractures in that they present with increased distance between the pedicles on anteroposterior (AP) plain films.

The Comprehensive Classification System was introduced by Magerl et al.[8,9] This classification involves three types, A, B, and C, each divided into three groups, each of which contains three subgroups. They represent a continuum of injury severity, with each step of the scale representing increasing instability. Type A injuries are compression injuries, which mainly involve the vertebral bodies. The mechanism of injury in type A is axial loading with or without flexion forces. These include compression and burst fractures and are the most common of the three types. The posterior soft tissues are not disrupted and any fracture of the posterior elements will be a vertical split that does not alter the stability of the injury. Group A1 are impaction injuries such as wedge fractures, which are the most commonly seen. A2 are split fractures through the vertebral body. A3 are burst fractures.

Type B injuries are distraction injuries and involve both the anterior and posterior elements. Type C injuries are multidirectional and involve both anterior and posterior element injury with rotation. These have the most significant instability and have the highest proportion of neurologic deficit. These injuries are discussed in detail elsewhere in this book.

One problem with these classification systems is poor reproducibility. A recent study by Wood et al.[10] had 19 different spine surgeons classify acute fractures of the thoracolumbar spine according to the AO and Denis classification systems. They found both systems to have only moderate interobserver and intraobserver reliability and repeatability. A prior study by Oner et al.[11] reported similar results. A good classification system would incorporate the mechanism of injury, radiographic appearance and degree of instability and guide in treatment decisions. Thus, future modifications to these classification systems and new systems should aim to improve this as well as guide treatment decisions.

A new classification system for thoracolumbar injuries was recently introduced by Vaccaro et al.,[12] named the Thoracolumbar Injury Classification and Severity Score. It is based on three injury characteristics: (1) morphology of injury determined by radiographic appearance, (2) integrity of the posterior ligamentous complex, and (3) neurologic status of the patient. Each of these main injury categories has subtypes with a numerical score associated with it. The scores for each of the three categories are added together to provide a comprehensive severity score. This score is then used to stratify patients into surgical and nonsurgical treatment groups. This system incorporates morphology as well as measures of instability to provide what may prove to be a very useful system that can facilitate clinical decision making. However, further studies on the validity and reliability of this classification need to be performed.

NEUROLOGIC INJURY

Neurologic injury occurs in 10% to 38% patients with thoracolumbar fractures.[13] Neurologic deficit must therefore be suspected in these types of traumatic injuries and full precautions taken. The entire spine should be immobilized with a cervical collar and backboard until clear. A full neurologic examination including a rectal examination and a check for the bulbocavernosus reflex is mandatory. Neurologic function should be assessed using a standardized method of evaluation, such as the Frankel or American Spinal Injury Association classifications. Neurologic deficits can be complete or incomplete cord injuries, mixed cord and root injury, and isolated root injury. Often sacral sparing is the only sign that a neurologic injury is incomplete. Also, spinal shock should be suspected, which may cause an incomplete injury to appear as complete with complete absence of reflexes, motor, and sensory function. Shock occurs at the time of injury and may persist for up to 72 hours. The end of spinal shock is characterized by the return of the bulbocavernosus reflex. Thus, the full extent of the deficit cannot be declared for a few days. Incomplete injuries have a better chance of some recovery because some of the structural continuity of the long spinal tracts is maintained. Complete injuries have almost no chance of recovery. The extent of neurologic recovery that occurs is related to the severity of

damage to the neural elements at the time of injury. If neurologic improvement is not seen within 24 hours, paralysis usually remains.[13]

INITIAL MANAGEMENT

Most patients sustain thoracolumbar trauma as a result of high-energy injuries such as motor vehicle accidents and falls from a height. A complete trauma evaluation is mandatory to detect concomitant life-threatening injuries. Often these patients sustain multiple injuries, as well as injuries at multiple levels of the spine. The entire spine should be evaluated in every trauma patient after the initial trauma protocols and resuscitation have been performed. Secondary spine injuries in a patient are often missed after detecting the first, more obvious injury, especially in patients who are unconscious or under the influence of alcohol and drugs. A complete history and detailed neurologic examination as discussed previously are necessary. A minimum of AP and lateral radiographs of the thoracolumbar spine should be taken, and a computed tomography (CT) scan should be performed if known or suspected fractures are present. A magnetic resonance imaging (MRI) can be useful if there is a neurologic deficit or to visualize ligamentous injury. The vast majority of patients with thoracolumbar fractures do not have neurologic deficits.

Although controversial, steroid protocols are commonly followed in the United States if there are no contraindications and it can be initiated within 8 hours of the injury in patients with neurologic deficits. A loading dose of 30 mg/kg of methylprednisolone should be given, followed by 5.4 mg/kg/hour for the maintenance dose. The maintenance dose is continued for 24 hours if the initial dose was given within 3 hours of injury or for 48 hours if the initial dose was given between 3 and 8 hours of injury. Patients treated with steroids may be at an increased risk of infection and gastrointestinal hemorrhage, so this must be considered.

Immobilization should be maintained throughout with protection of the neural elements until definitive treatment can be administered. Finally a careful analysis of several factors, including mechanism of injury, spinal stability, neurologic status, anatomy, body habitus, medical comorbidities, time of injury, and radiographic studies, must be done to determine the most appropriate treatment plan.

TREATMENT CONSIDERATIONS

The primary goals of treatment in patients sustaining spinal trauma are to provide neural protection and optimize neural recovery, restore and maintain anatomic spinal alignment and stability, maximize function and minimize pain, and reduce morbidity and mortality to provide the best possible outcome for the patient. To achieve these goals, the decision-making process in the treatment of thoracolumbar fractures operatively or nonoperatively involves the evaluation of many variables. Factors that must be considered include the mechanism of injury, degree of stability of the fracture and adjoining segments, the presence or absence of neurologic deficits, whether neurologic deficits are progressing or stable, and the degree of deformity and chance of progressive deformity. Other factors include concomitant injuries and patient comorbidities, such as patient ability to tolerate a cast or brace, morbid obesity, the presence of chest tubes, and contraindications to immobilization.

The majority of spine fractures do not require surgery. Almost all compression fractures and many burst fractures can be treated nonoperatively. However, certain situations exist in which surgical treatment is almost always recommended. These include deteriorating neurologic status or progressive deformity despite appropriate nonoperative treatment. In situations in which no stability with healing can be expected, such as complete ligamentous disruption or dislocation without bony contact, surgery is also usually required.

One of the most critical steps is to decide if the fracture is stable or unstable. There is no universally accepted definition of stability, and thus many factors must be considered in assessing stability. Denis[5] essentially defined instability as a disruption of two or three of the spinal columns, with the middle column being key to stability. The middle column is considered to be critical in assessing stability and in differentiating a compression fracture from a burst fracture. Denis categorized the instability as neurologic, mechanical, or both. Neurologic deficit in the setting of a spinal fracture constitutes neurologic instability. Mechanical instability involves fracture patterns with disruption of two or more columns, especially with distraction of the posterior elements, which can lead to further kyphotic deformity. However a weakness of this definition is that many burst fractures are stable and can be treated nonoperatively.

A study by James et al.[14] evaluated the relative contributions of the anterior, middle, and posterior columns to spinal stability using a human cadaver L1 burst fracture model. They found that the stability of the posterior column, rather than the middle column, was the best indicator of fracture stability. They also noted healing without deformity in a series of patients with intact posterior columns but varying degrees of anterior and middle-column deformities. They proposed that the classic burst fracture with only anterior and middle column injury is stable and can be treated nonoperatively in the absence of neurologic deficit.

COMPRESSION FRACTURES

The vast majority of compression fractures can be treated by nonoperative means. They generally involve only one column and are stable with a very low occurrence of neurologic injury. Surgical consideration may be warranted if there is greater than 50% loss of height of the anterior vertebral body or greater than 25 to 30 degrees of kyphosis. In such

fractures, a CT scan should be considered to evaluate the middle column for a subtle burst fracture not apparent on plain films. As the posterior elements may be damaged with this degree of deformity, an MRI may be useful to identify damage to the ligaments and to differentiate benign from malignant compression fractures.[15]

When a compression fracture is treated nonoperatively, it can usually be done with a molded hyperextension orthoses, such as a thoracolumbarsacral orthoses (TLSO), or a hyper-extension cast for severe fractures. The patient may be weight bearing as tolerated in the brace and with treatment similar to that for burst fractures as mentioned in the next section.

If surgical management is needed for unstable compression fractures, a posterior approach is usually adequate. This is because, by definition, the middle weight-bearing column is intact and the deformity indicates a disruption of the posterior elements. Fixation can be accomplished with segmental hook-rods or pedicle screws-rods with distraction and extension forces. A lateral compression fracture may be stabilized with a compression construct on the non-injured side and a distraction construct on the injured side.

BURST FRACTURES

Although most compression fractures can be treated nonoperatively, the decision to treat burst fractures operatively or nonoperatively is perhaps the most controversial of all the spine fracture types. Even when a surgical decision is made, the best approach is controversial, as discussed in full in the next section. The main determining factors to consider are the stability of the fracture, the neurologic status, degree of angulation and the amount of deformity present. Common recommendations are that greater than 20 to 30 degrees of kyphosis, greater than 50% loss of vertebral body height,[16] subluxation of the posterior facets, increase in interspinous process distance, or neurologic deficits indicate instability and that such patients would benefit from surgery. Most of the previous are signs of posterior ligamentous disruption. Many sources cite a spinal canal compromise greater than 50% to indicate instability and the need for surgery. A study by Meves and Avanzi[17] found the probability of neurologic injury to increase with increasing amounts of canal occlusion. However the percentage of spinal canal compromise correlates poorly with neurologic deficit, prognosis, and clinical outcome according to many other studies.[18–23] Thus, because of the ongoing controversy in the literature, it cannot be routinely recommended as a primary criterion for surgery in the absence of a neurologic deficit.

The reason for the poor correlation may be that most of the neurologic injury may occur at the time of impact and the position of the fragments at the time of evaluation may not represent the severity at impact.[24,25] Wilcox et al.[25] performed a dynamic study using calf spines and a finite element model and found that the maximum cord compression and canal occlusion occur at the moment of impact. The bone

fragments were then recoiled by the posterior longitudinal ligament and intervertebral disk attachments to their final resting position. Thus, the final position of the fragments on subsequent imaging probably does not correlate to injury severity and risk. The neurologic deficit may result from a combination of factors such as the initial forces at the time of injury on the neural elements as well as associated edema, hematoma, and ischemia.

In addition, the spinal canal undergoes significant spontaneous remodeling after injuries, with resorption of retropulsed bone fragments.[26–33] Several studies have confirmed this and remodeling appears to continue until the canal area reaches the normal range.[31] Some authors have therefore concluded that removal of intraspinal fragments is not necessary in neurologically intact patients.[31,33] Remodeling occurs in patients treated both operatively and nonoperatively and is used as an argument for nonoperative treatment.

Most authors tend to recommend operative treatment in thoracolumbar burst fractures with any neurologic deficits or instability. If a patient treated nonoperatively experiences neurologic deterioration, then surgical treatment is usually recommended. However, even these are not universal recommendations. A nonsurgical approach for patients with a neurologic deficit is rarely recommended but may be indicated in patients with a complete spinal cord injury and other medical contraindications to surgery. The most controversy involves the treatment of burst fractures without neurologic deficits.

Several studies have compared operative versus nonoperative treatment of thoracolumbar burst fractures. However, direct comparison of the results of such studies is limited as the studies involve injuries of varying types and severity. More severe injuries are usually more likely to receive operative treatment and this must be considered in evaluating any results.[34] Not all studies look specifically at burst fractures and some lump different types of fractures into one group. Perhaps one of the most difficult factors in comparing studies and results is the lack of a clear consensus regarding what constitutes a stable fracture. Published studies do not all have the same inclusion and exclusion criteria for stability. Many of the studies define stable fractures as having no substantial posterior ligamentous injury, but several studies found successful results in patients with burst fractures and posterior ligamentous disruption treated nonoperatively.[35,36] On the contrary, Denis et al.[7] performed a retrospective analysis on 52 patients with thoracolumbar burst fractures in the absence of neurologic deficit. Of the 39 patients in the nonoperative group, 17% developed neurologic problems and 25% did not return to full-time work. All patients treated surgically and who had no related disability returned to work full time. It was concluded that surgical treatment was the best option for these types of burst fractures.

Surgical treatment of all burst fractures is, however, not routinely warranted. Several authors have not found such a high rate of deterioration in patients treated nonoperatively.

Mumford et al.[20] experienced only a 2.4% incidence of neurologic deterioration in 41 burst fractures without neurologic deficit treated nonoperatively. Likewise, others have found nonoperative treatment to be highly effective for these types of injuries without neurologic deficits in comparison with surgical treatment. Wood et al.[23] performed a prospective, randomized study comparing operative (anterior or posterior fusion and instrumentation) versus nonoperative (application of a body cast or orthosis) treatment in patients with thoracolumbar burst fractures without neurologic deficit. Forty-seven patients without neurologic deficit were randomized into one of the two groups. They excluded unstable fractures with suspected or known posterior osteoligamentous disruption. Average follow-up was 44 months. No statistical difference was found between the two groups with respect to functional outcome and return to work, kyphosis, or pain. They concluded that in such patients, surgical treatment provided no major long-term advantage compared with nonoperative treatment. Shen et al.[37] prospectively compared the results of short segment posterior fixation versus nonoperative treatment in 80 patients without neurologic deficit or posterior arch injuries. The 33 patients in the operative group had better outcome scores at 3-month follow-up, but outcomes were similar at 6 months and beyond. Kyphosis correction was initially better in the surgical group, but this advantage was also lost with time. No neurologic deficits developed. Likewise, many other studies find nonoperative treatment to be successful in burst fractures without initial neurologic injury.[21,35,38,39]

A review of the literature strongly supports that nonsurgical treatment, usually consisting of bracing and early mobilization, is effective for stable thoracolumbar burst fractures without neurologic deficit. In patients with the poorly defined criteria for instability and in those patients with neurologic deficits, operative treatment is usually recommended. Early mobilization, short-term functional results, and correction of kyphosis seem to be the main benefits of surgical treatment. However, the improved correction of kyphosis has not correlated well with clinical outcomes and long-term functional results are similar. Surgical treatment carries an increased risk of infection, bleeding, iatrogenic injury, and cost. Ultimately, because of the lack of a clear consensus regarding the treatment of burst fractures without a neurologic deficit, the individual surgeon must assimilate all the factors involved in the case and use his or her own judgment to determine the plan of care.

NONOPERATIVE TREATMENT

Once the decision is made to treat a compression or burst fracture nonoperatively, the treatment plan must be devised. Historically, the earliest treatment for these fractures was nearly always nonoperative, with the majority treated with bed rest, immobilization, and plaster body casts. Bed rest, although effective, carries the risk of severe complications such as deep vein thrombosis and skin breakdown. Recent trends have therefore swayed toward early mobilization. The
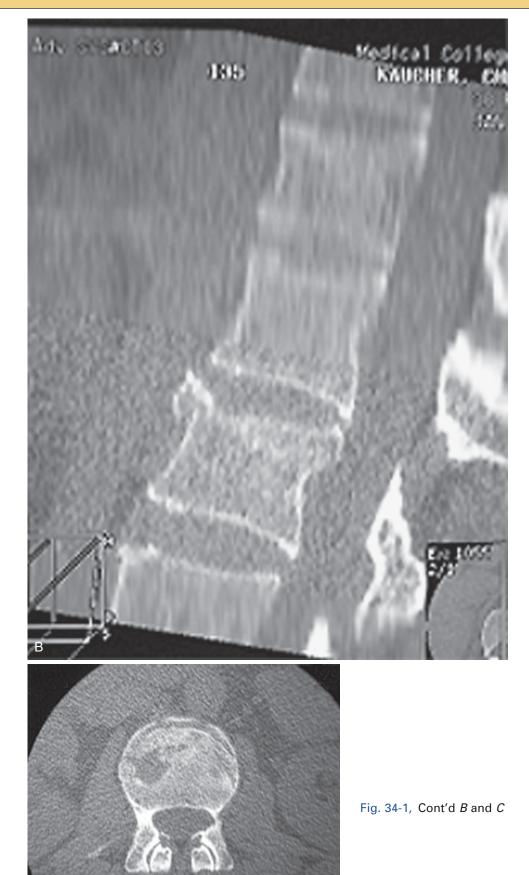
majority of patients can be successfully treated by nonoperative means with the help of a hyperextension cast or a well-molded orthoses. Even severe burst or compression fractures that are unstable may be treated in this fashion.

Tropiano et al.[40] reported that closed reduction and casting was a safe and effective treatment for thoracolumbar burst fractures in a retrospective study of 41 neurologically intact patients. Reduction was performed on a Cotrel traction frame. Hyperextension casting can generate significant hyperextension forces to maintain sagittal alignment, but its application is cumbersome and time consuming. Casting is, however, inappropriate in polytrauma patients with chest or abdominal trauma or bowel problems such as an ileus. Moreover, it tends to be less comfortable and personal hygiene is difficult. Hyperextension cast may therefore be more appropriate when the injuries are severe and molded orthoses cannot provide the same degree of stabilization, or when patient compliance is questionable.

Orthosis is usually the preferred vehicle for nonoperative treatment of almost all compression and most burst fractures (Fig. 34-1). Initially, bed rest with spine precautions should

Fig. 34-1 Lateral radiograph *(A)* and sagittal CT reconstruction *(B)* of a patient with compression fracture. Axial images show intact middle column and spinal canal. This patient was successfully treated in a TLSO.

Fig. 34-1, Cont'd *B* and *C*

be maintained until the custom-molded TLSO is fit and in place. An experienced and skilled orthotist is necessary for success with these orthoses. The patient can then get out of bed to chair and begin ambulation with assistance. Physical therapy is also initiated in the first few days for extremity range of motion and exercises.

Standing or sitting AP and lateral radiographs in the brace should be taken once a patient is able to cooperate (Fig. 34-2). A study by Mehta et al.[41] compared supine and erect radiographs in 28 patients with thoracolumbar fractures suitable for nonoperative treatment. With the weight-bearing films, the Cobb angle and anterior vertebral compression increased in comparison to the supine films. This finding on the erect films was enough to change the management in 25% of the patients, who subsequently underwent surgical stabilization.

If alignment is maintained on the erect weight-bearing films, ambulation may continue and the patient can be discharged when he or she is capable of activities of daily living and is otherwise stable. The brace must be maintained at all times and close follow-up is warranted with standing radiographs at each visit to ensure alignment is maintained.

Patients should be made aware of the symptoms of cauda equina syndrome or neurologic deterioration. If neurologic deterioration or progressive deformity occurs, nonoperative treatment has failed and surgery is likely necessary. If there is no adverse event, immobilization is usually continued for approximately 3 months. Once healing is evident, flexion and extension radiographs out of the brace or cast are taken and if the fracture and alignment appear stable, the brace can be discontinued.

OPERATIVE TREATMENT

Whether to perform an anterior, posterior, posterolateral, or combined anterior and posterior approach for the treatment of thoracolumbar burst fractures remains a controversy, and each approach has its own advocates. Adequate decompression and restoration of spinal stability are key components of surgical planning. With no clear standard of care, the decision must therefore be made based on the type of fracture, surgeon experience, and familiarity with a certain approach, with these goals in mind.

ANTERIOR APPROACH

Advocates of the anterior approach cite that it allows direct visualization of the anterior thecal sac for direct decompression and allows for reconstruction of the major weight-bearing portion of the spine. The anterior column translates approximately 80% of the axial load through the spine, although in the lower lumbar spine, more weight is carried through the posterior elements. An anterior approach is thus useful for treating anterior neural compression, such as occurs in burst fractures (Fig. 34-3). Anterior instrumentation

Fig. 34-2 Upright lateral radiograph in a TLSO *(A)* and axial CT *(B)* showing significant loss of height, involvement of middle column and small amount of retropulsion in a neurologically intact person. This patient was also treated conservatively.
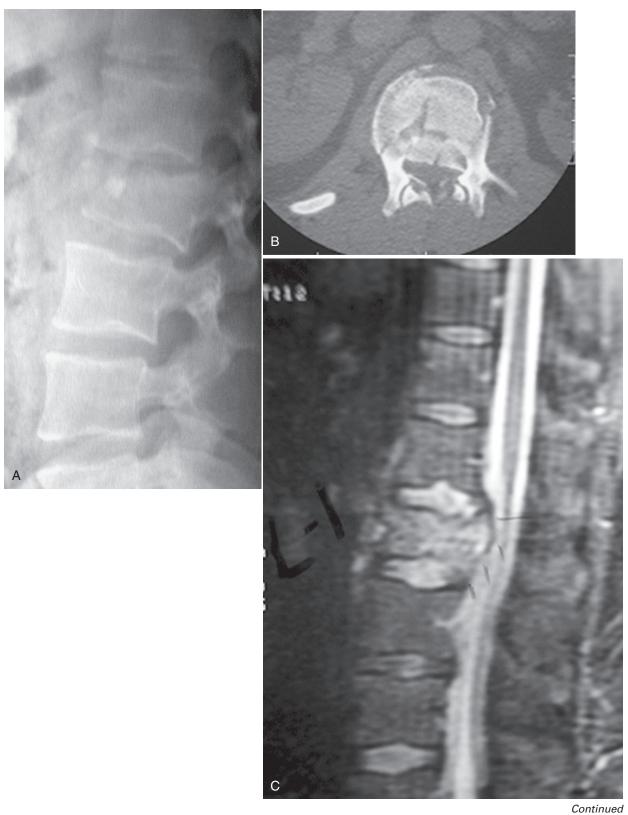
Fig. 34-3 Lateral radiograph *(A),* axial CT *(B),* sagittal and axial MRI (*C* and *D*) images of a patient with burst fracture and significant canal compromise was treated with corpectomy and cage reconstruction through an anterior approach. Postoperative anteroposterior and lateral radiographs are shown in *e* and *f.*

Fig. 34-3, Cont'd *D, E* and *F*

can be beneficial in unstable burst fractures to prevent late kyphotic collapse and deformity. However, it is more difficult to correct kyphosis with an anterior-only approach. It is most appropriate in patients with significant neural compression and deficits, especially if they have minimal kyphotic deformity, and an absence of significant posterior osteoligamen-tous disruption. If extensive posterior disruption is present, anterior-only fixation may result in instability and splaying of the posterior elements.

Esses and associates[42] conducted a prospective, randomized study of 40 patients with acute thoracolumbar burst fractures that were treated with posterior distraction using

pedicle instrumentation or anterior decompression and instrumentation. The extent of canal compromise reduced from 58% preoperatively to 4% postoperatively with the anterior approach and from 44.5% to 16.5% with the posterior approach, a significant difference in the degree of canal clearance between the two approaches. There was not a significant difference in kyphosis correction between the two groups. The authors concluded that, although posterior distraction instrumentation can effectively decompress the canal and correct kyphosis in patients sustaining burst-type injuries, anterior surgery results in a more complete and reliable decompression of the canal.

Kaneda et al.[43] managed 150 consecutive patients with thoracolumbar burst fractures and associated neurologic deficits with single-stage anterior decompression, strut grafting, and instrumentation and reported excellent results. Ninety-five percent of the patients had at least one Frankel grade improvement in neurologic function. Fusion rate was 93%, and 86% of patients were able to return to their previous occupations. Bradford and McBride[44] found greater neurologic recovery in thoracic and lumbar fractures with incomplete neurologic deficits treated by anterior decompression than those treated by posterior decompression. Wood et al.[45] performed a prospective, randomized study comparing anterior versus posterior fusion with instrumentation of thoracolumbar burst fractures without neurologic deficit and reported similar outcomes with the two approaches, although there was a trend toward fewer complications with the anterior approach. Several other authors also have reported good results with this approach.[46–49]

Some of the other advantages of an anterior approach include availability of local corpectomy bone and resected rib for bone grafting, thereby obviating the need for iliac crest bone grafting. Additionally, only three vertebrae need to be fused with an anterior approach, unlike a posterior approach, which usually requires inclusion of two vertebra above and two below the level of the injury.

Disadvantages of the anterior approach include the extensive approach, vascular complications, pain and morbidity from the thoracotomy, and pulmonary complications. A retrospective review by Oskouian and Johnson[50] on 207 patients who underwent anterior thoracic and lumbar spinal decompression and reconstruction found the following incidence of vascular complications: In seven patients (3.4%), direct vascular injuries developed as a result of surgical techniques or error; one patient died as a result. Five patients (2.4%) developed deep venous thromboses, and one patient in this subgroup died of pulmonary embolism.

In recent years, there has been a trend toward minimally invasive thoracolumbar spine surgery with a view to minimize morbidity associated with extensile approaches. Standard thoracoabdominal approaches also produce significant denervation of the abdominal wall musculature. Minimally invasive thoracoscopically assisted approaches have been developed which facilitate access to the retro-

peritoneal space down to the L3 vertebra with transthoracic detachment of the diaphragm, if necessary. This surgical approach exploits the anatomical peculiarity of the lowermost attachment of the diaphragm, because the perpendicular projection of the costodiaphragmatic recess corresponds with the level of the L2 vertebra. Such an exposure dramatically reduces morbidity, while providing excellent visualization and working space for decompressive procedures such as corpectomy and surgical stabilization of the thoracolumbar junction.[51]

ANTERIOR DECOMPRESSION

The approach is usually performed from the left side. The patient is carefully turned to the lateral position and secured with appropriate padding of all pressure points. The table is jack-knifed to improve access and to open up the space between the costal margin and the iliac crest. Neurophysiologic monitoring is recommended. Unless the surgeon is familiar with the extensile approach or with a minimally invasive approach, a vascular and cardiothoracic surgeon typically performs the exposure. Typically the rib two levels above the fracture are removed and the diaphragm is divided after stay sutures have been placed. Segmental vessels are ligated and exposure of one vertebra above and below the fracture is performed after radiographic confirmation of the fracture site. The psoas muscle often gets in the way of exposure at L2 and below. It is helpful to secure the left hip in slight flexion during positioning, which facilitates easier retraction of the psoas muscle. Nerve roots are typically located in the posterior half of the muscle and should be carefully protected. Care is taken to ensure that vessels are protected anteriorly. The posterior extent of the vertebra can be located by placing a Penfield elevator in the intervertebral foramen. The ipsilateral pedicle may also need to be resected to locate the posterior extent of the spinal canal. Proper orientation throughout the procedure is mandatory to avoid violation of the spinal canal and to decrease the risk of vascular injury.

Initially, diskectomies are performed at the level above and below the fractured vertebra. Corpectomy is typically begun with a large rongeur and a large part of the vertebra can be quickly removed, and the resected bone saved for grafting. The far lateral and anterior cortices are left intact, and a large trough is thus created. Decompression of the retropulsed fragments is performed next. The posterior cortex is thinned initially with a high-speed bur and the space between the retropulsed fragments and posterior longitudinal ligament is developed. Retropulsed fragments are then peeled away from the spinal canal with the help of angled curets. Decompression is begun at the level of the contralateral pedicle and continued all the way across to the ipsilateral pedicle to ensure complete removal of retropulsed fragments. As decompression progresses from right to left, the dura starts bulging into the trough without obscuring visibility of the retropulsed fragments. Kyphosis can be partially

corrected by applying anteriorly-directed manual pressure. Sizing of the cage and instrumentation can then proceed as discussed in a subsequent section.

POSTERIOR APPROACH

A posterior approach for the treatment of thoracolumbar burst fractures relies on indirect decompression through ligamentotaxis rather than direct decompression. Gentle distraction through instrumentation is used to decompress the neural canal. This mechanism is dependent on an intact posterior longitudinal ligament and even more so on remaining annular fibers that originate in the annulus of the superior vertebra in the midportion of the endplate and insert into the lateral margins of the retropulsed fracture fragment.[1] This indirect mechanism can result in significant decompression of the spinal canal.[52–54] A transpedicular approach in the lumbar spine or a posterolateral approach in the lower thoracic spine may facilitate decompression by impacting the retropulsed fragments. A posterior approach thus allows for adequate decompression in many cases, although a second stage, anterior surgery may sometimes be needed when there is severe anterior and middle-column injury, as is common in burst fractures. Advocates of a posterior approach cite good results, experience, familiarity with the anatomy, a decreased need to rely on an approach surgeon, and early mobilization as advantages.

Disadvantages include an inability to directly decompress the canal unless large portions of the neural arch are resected. The neural elements are at risk as they lie between the fracture fragments and the surgeon. If there is deformity of the anterior or middle columns, which is often the case with burst fractures, recurrence of the deformity or instrumentation failure may occur after posterior-only fixation as the anterior column is not restored.[55–58] Harvesting of iliac crest bone is often necessitated with a posterior approach because of a lack of availability of local autograft bone for grafting. A posterior approach also requires fusion across four or more segments.

COMBINED APPROACH

A combined approach can also be used, either in a staged fashion or simultaneously. When a posterior approach and reduction alone fails to provide adequate neural decompression, resulting in a persistent neurologic deficit, a secondary anterior surgery is indicated. A combined approach can also be considered if there is severe circumferential spinal canal compromise and cord compression. However, it is most useful in fracture dislocations and severe three-column injuries rather than burst fractures. Dimar et al.[59] treated 84 burst fractures with anterior decompression, autologous iliac crest strut graft, and posterior instrumentation and fusion and reported good results with this combined approach. Rarely, when burst fractures involve contiguous vertebrae, staged anterior followed by posterior surgical intervention is needed

(Fig. 34-4). When a determination has been made that a combined staged approach is necessary, it is beneficial to place anterior screws in the lower half of the vertebral body, so that they would not come in the way of subsequent pedicle screw insertion from a posterior approach. If a posterior approach is performed first with transpedicular instrumentation, there is usually sufficient room for placement of anterior screws in the lower half of vertebrae through a subsequent anterior approach.

It should be noted that laminectomy alone should never be used for the treatment of burst fractures. A laminectomy cannot adequately decompress the spinal canal and can destabilize the spine even more. It can, in fact, be associated with an increase in neurologic deficits.[60,61]

Generally, it can be recommended that an anterior approach be used when there is a large retropulsed bone fragment in the spinal canal with a severe neurologic deficit. Posterior indirect or posterolateral decompression is recommended for burst fractures with minimal or no neurologic deficits and with burst fragments that are relatively small and located posterolaterally. If there are nerves entrapped between lamina fractures, a posterior approach is also required. In cases in which the fractures are subacute (greater than 3 to 4 weeks old), an anterior approach is required.

INSTRUMENTATION

Many different types of instrumentation systems exist and the appropriate one for a given fracture depends on a variety of factors. All the varying instrumentation systems have relative strengths and weaknesses. The choice of which instrumentation system to use depends on the fracture type and spinal level, whether it is for anterior or posterior fixation, and the surgeon's experience and preference. Nonsegmental hook-rod (i.e., Harrington rods) and rod-sleeve systems are of historical interest only, and the major instrumentation options for posterior fixation include segmental hook rods or short segment pedicle screw constructs. The anterior instrumentation options include plates or rods with screws as well as varying struts and vertebral body replacements. Because burst fractures are primarily caused by axial loading and flexion forces, systems that impart distraction and extension are optimal. It is important to avoid systems with a compression construct because these can further cause retropulsion of fracture fragments into the spinal canal.

The basic goals of using instrumentation are to achieve anatomic reduction and stable fixation of the fracture and to provide neural decompression when necessary. There are many varying opinions in regard to the number of spinal segments to fuse, the number of segments to instrument, the configuration of hooks and pedicle screws, as well as whether or not to remove the instrumentation. An in depth review of these topics are beyond the scope of this chapter. following is a brief description is instrumentation type and their potential use for thoracolumbar burst and compression fractures.

HOOK-ROD SYSTEMS

Although Harrington rods were one of the first effective instrumentation systems used for thoracic and lumbar spine fractures,[61–66] they are usually not appropriate for burst fractures. Harrington rods are nonsegmental rod-hook systems introduced in the 1960s. Although they can provide spine stabilization and allow early mobilization, they have little intrinsic stability and mainly have a role only in the thoracic spine. They can provide only semirigid fixation. Likewise, Luque instrumentation systems cannot provide adequate axial stability to treat thoracolumbar burst fractures and should not be used. Another disadvantage of these nonsegmental hook-rod systems is that they usually extend two to three
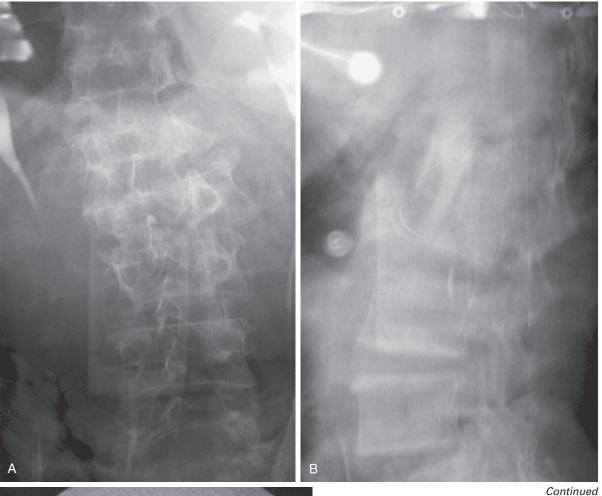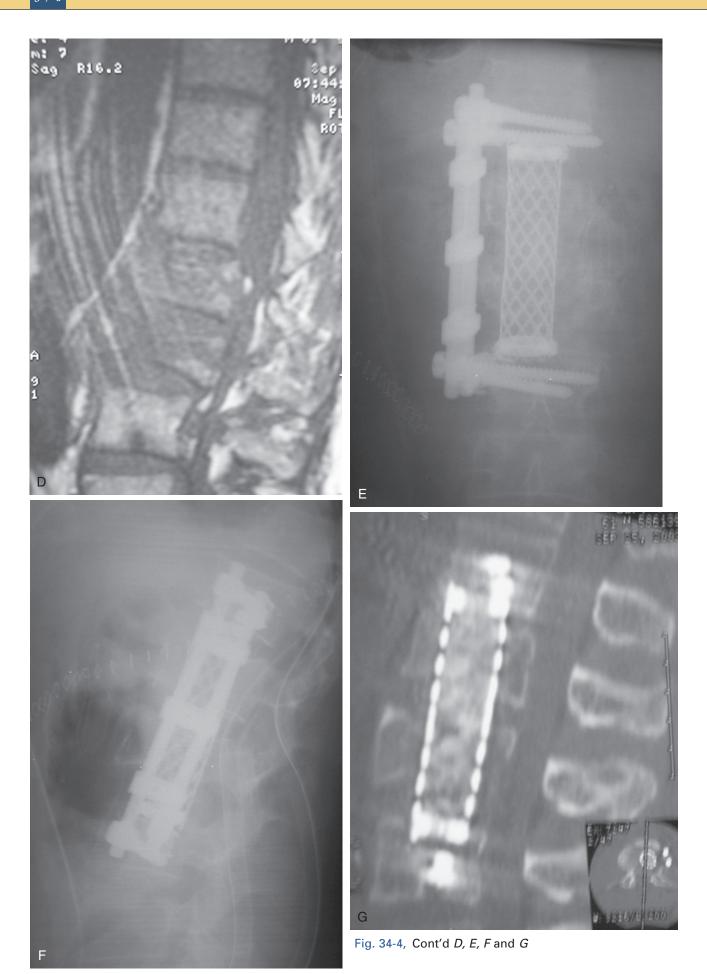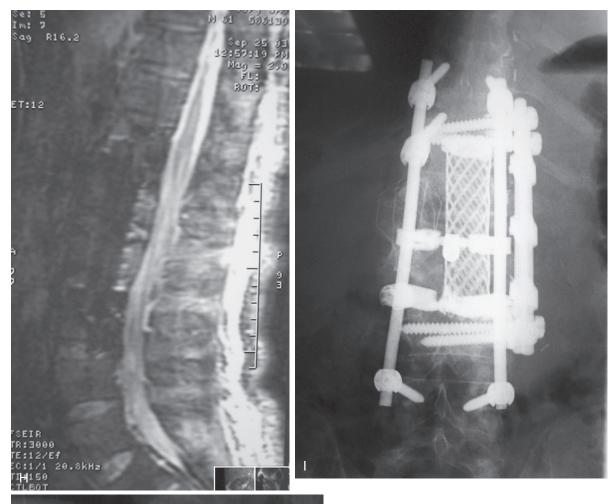
Continued

Fig. 34-4 Anteroposterior and lateral radiographs *(A, B)* of a patient with severe burst fracture of L2 and L1 vertebrae and incomplete neurologic injury. Axial CT through L2 burst fracture *(C)* and sagittal MRI *(D)* images. Corpectomy of L2 with spinal canal clearance and a channel corpectomy of L1 was emergently performed along with placement of a Harms cage and T12-L3 anterior Kaneda instrumentation. *E–H,* Immediate postoperative anteroposterior and lateral radiographs, with sagittal CT reconstruction and T2 sagittal MRI images showing satisfactory decompression of the spinal canal. Two days later the patient underwent posterior stabilization (*I* and *J*). He made full neurologic recovery.
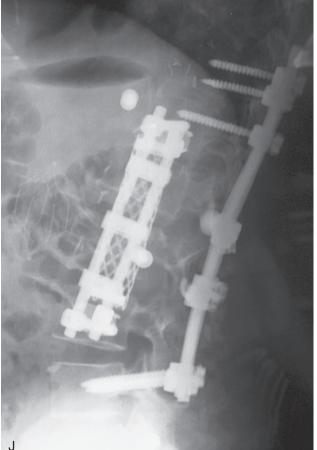
Fig. 34-4, Cont'd *D, E, F* and *G*

Fig. 34-4, Cont'd *H I* and *J*

levels above and below the injury. They can also be prone to breakage[67–69] and hook dislodgement. The distraction force they provide can result in kyphotic forces to the lumbar spine that can lead to loss of lumbar lordosis and flat back deformity, which can be quite painful. Additionally, hook-rod systems are typically associated with lower stiffness and often lead to loss of correction and recurrence of 3 to 12 degrees of kyphosis.[70]

Traditional Harrington rod constructs can only apply a reduction force in a single plane, either axial distraction or compression. To decrease the potential for flat back deformity, several adjustments can be made. By contouring the rods in a sagittal plane or adding sleeves, such as in the Edwards instrumentation system, both a distraction and extension force can be applied, with individual adjustments of each force vector. Edwards and Levine[71] reported good results with this system, with a significant reduction in kyphosis and an increase in vertebral body height and spinal canal patency. However, these systems have, in large part, been superseded by newer segmental instrumentation systems.

SEGMENTAL INSTRUMENTATION

For burst fractures, segmental fixation systems with multiple hook or screw fixation is generally a decent option for posterior instrumentation.[72,73] Hooks alone, however, necessitate inclusion of several segments in the construct to achieve even a reasonable degree of stability. Rod long and fuse short strategies have tried to address concerns that arise from multilevel fusion. However, this technique requires additional surgery f or subsequent removal of the rod, and the immobilized but unfused segments are still at risk of developing arthritic changes with ensuing pain and stiffness. Short-segment instrumentation can provide sagittal, axial, and torsional stability in the lumbar spine which is superior to nonsegmental hook-rod systems or sublaminar wiring.[72,73] Pedicle screws may be used alone or in combination with hooks, and these systems can be used in both distraction and compression to correct complex spinal deformities and maintain normal sagittal contours. Pedicle screws can provide rigid three-column fixation and stability, provide four-point fixation, and allow for immobilization of fewer spinal segments. Stronger corrective forces can be applied than with other systems. They do not depend on an intact posterior neural arch and thus can be used in patients with lamina and posterior element fractures. The degree of distraction required is not as great as with nonsegmental systems, and thus the risk of flat back deformity is lessened.

There are several disadvantages to these systems as well. First of all, pedicle screw placement may be difficult in the thoracolumbar spine, especially if the pedicles are small. Pedicle screw placement in this region of the spine is surgeon-and experience-dependent. Moreover, a high rate of screw failure has been reported in treating unstable spine fractures with short segmental fixation.[34,58,74,75] It is important to consider the biomechanical implications of treating severe burst fractures with posterior instrumentation. With severe burst

fractures, there is a failure of the anterior and middle columns. If these columns are not able to help share and support the axial load, a bending moment will occur in the posterior instrumentation. The anterior compressive forces must be completely absorbed by the posterior instrumentation.

Posterior instrumentation requires sacrificing of more segments than does an anterior approach. Gurr et al.[72] performed an in vitro study on calf spine and noted that a three-level anterior instrumentation with a Kaneda device produces a similar degree of stiffness as a five-level posterior pedicle screw construct. A biomechanical study by Slosar et al.[76] demonstrated that posterior instrumentation is not in an appropriate position to withstand these forces. This can result in instrument failure and bending and an inability to maintain adequate deformity correction in the sagittal plane. Once this initial insult has occurred, progressive deformity and collapse is likely.

Duffield et al.[77] performed a mechanical study of the effects of implant stiffness on load sharing and stress shielding, of vertebral column load sharing on implant fatigue life and of instrumenting one versus two levels adjacent to a comminuted segment on implant internal loads using finite element models. They concluded that single-level posterior element instrumentation adjacent to a comminuted spine segment will have a finite fatigue life and that the internal bending moments within an implant can be reduced to levels with a low probability of causing implant fatigue if anterior column support equivalent to a healthy motion segment is present. Instrumentation two levels adjacent to a comminuted segment as opposed to one level can reduce the flexion bending moment in the implant. Gaines et al[78] and McCorack et al[79] demonstrated that comminuted vertebral body fragments do not transfer load as well as intact vertebrae. As such, short-segment posterior fixation of comminuted burst fractures with displaced fragments was more likely fail. The load-sharing classification is based on these findings.[79] These studies demonstrate the importance of creating load sharing in these types of fractures. Thus, if load sharing cannot be obtained with posterior fixation alone, anterior instrumentation may be needed. For burst fractures in the low thoracic or upper lumbar spine, if posterior short segment fixation is used, a second-stage anterior approach may be needed depending on the weight-sharing property of the anterior vertebral body fracture.

ANTERIOR INSTRUMENTATION

Anterior corpectomy and reconstruction with instrumentation allows for direct restoration of the weight-bearing columns of the spine. Modern anterior instrumentations with interbody grafts have been found to restore stability in all motion planes in unstable fractures.[80] An et al. found this fixation to be more rigid in flexion and extension than fixation with pedicle screws alone. Addition of an interbody graft also provided additional stability to posterior constructs.[81] Fewer levels can typically be fused in comparison with posterior fixation and good results have been reported in clinical and biomechanical studies.[43,46,49,70,72,80–84] Anterior instrumentation devices primarily

serve as load-sharing devices and must provide support for the anterior and middle columns. They include both options for corpectomy reconstruction, such as autogenous or allograft bone grafting and titanium or carbon fiber mesh cages and screws with plates or rods for anterior fixation.

Interbody reconstruction following corpectomy can be done with bone graft from the iliac crest, rib, or fibula. The sizing and shaping of these grafts is important for optimal results and they are associated with the typical donor site morbidity. Titanium, reinforced carbon fiber, or PEEK cages are therefore preferred for reconstruction of corpectomy defect. Added stability and fixation can be gained using an anterior stabilizing plate or rods. Expandable cages are now available, which facilitate easier insertion of the cage following corpectomy and more controlled restoration of the spinal column height.

Modern anterior instrumentation devices such as screw rod constructs and screw plate systems are used to stabilize the anterior and middle column following cage placement in the corpectomy defect. They typically consist of anterolateral screw fixation along with longitudinal plates or rods. They can provide stability while sharing axial loads with a strut graft or cage. Vertebral screws should be directed in a triangular pattern to achieve maximum resistance to pullout forces. Posterior screws are typically bicortical, and care should be taken to ensure that screws do not violate the spinal canal. The screws are inserted in conjunction with a plate or rods. Plates such as Z-plates are lower profile, but screw rod constructs tend to be more versatile and are usually well-tolerated despite being more prominent than a plate. The Kaneda device and variations thereof are commonly used anterior systems that have shown good results.[43,84] Such a construct is very rigid and consists of screws and paravertebral rods that allow distraction during spinal decompression and then compression of implanted grafts.

Typically if an anterior approach is being done as a second stage following posterior fixation, corpectomy and interbody reconstruction is sufficient without the need for anterior instrumentation. An et al.[85] found pedicle screw instrumentation to provide rigid stabilization, which is superior to hook-rod strategies, in the posterior stabilization of unstable burst fractures with a corpectomy anterior defect in a calf spine model. Therefore posterior instrumentation may be sufficient along with interbody reconstruction. However, if a sole anterior approach is used, dual rods or low-profile plating instrumentation is generally recommended as well. Anterior-only instrumentation can be used only in the absence of significant posterior osteoligamentous disruption as discussed in previous sections of this chapter. An anterior-only construct is more likely to fail in the presence of posterior disruption without posterior instrumentation.

COMPLICATIONS

Thoracolumbar fractures treated both operatively and non-operatively can have significant complications. Of course the worst complication is death, of which thromboembolism is a common cause in patients surviving the initial injury, especially in patients with complete neurologic deficits. Neurologic deficits are one of the most feared complications. The risk of complications such as deep vein thrombosis, atelectasis, pneumonia, and decubitus ulcers can be reduced with early mobilization. Urinary tract infections are common if neurologic deficits exist and intermittent catheterizations should be instituted in place of indwelling catheters as soon as possible. Chronic pain is also a problem.

In patients treated surgically, additional risks include blood loss, infection, and technical complications. Trauma patients experience an infection rate nearly three times that of patients undergoing elective spinal surgeries.[86] Deep spinal infections occur in 3% to 10% of patients with spinal instrumentation in most reported series.[13] Acute infections should be treated with serial irrigation and debridements and long-term antibiotics. Hardware should be left in with early acute infections if it is stable to allow for fusion. In late acute fractures in which the fusion is established, the hardware should be removed. Technical complications can relate to instrumentation failure, the use of unsuitable instrumentation for a particular fracture, or overdistraction or compression. This can lead to instability and deformity progression and improper instrumentation placement can lead to vascular erosion or placement into the spinal canal. There are also complications related to the different approaches, as discussed previously.

REHABILITATION

Perhaps the most important component of early rehabilitation in patients with traumatic spinal injuries is early mobilization to lower the risk of deep vein thrombosis, atelectasis, pneumonia, decubitus ulcers, and other associated complications of immobilization. Patients with spinal cord injury will require a much more extensive and lengthy rehabilitation. Early passive range-of-motion exercises and appropriate splinting is necessary to prevent contractures in such patients. Bowel and bladder management programs are also necessary. Physical and occupational therapy should be daily while the patient is in the hospital. Nutrition must be optimized to decrease the risk of infection and promote bone healing. Tobacco use must be discouraged. Finally, social and occupational support is needed for patients' activities of daily living and return to society.

CONCLUSION

Thoracolumbar fractures can be devastating injuries that result in significant patient morbidity and mortality. Surgical treatment of burst fractures has advanced immensely in the past few decades, but the majority of these fractures can still be treated nonoperatively. If neurologic deficits are present or progress, or if the fracture is unstable, surgical intervention is usually warranted. The surgical approach

remains controversial and both anterior and posterior approaches can be effective. However, recent trends are favoring an anterior approach with corpectomy and instrumentation to reconstruct the weight-bearing anterior spine that is deficient in burst fractures. Posterior instrumentation is necessary with posterior disruption.

In the future, studies may aim to better delineate stable from unstable fractures, further assess the value of surgery in neurologically stable patients, develop guidelines for the surgical approach and instrumentation to be used, assess long-term follow-up on these patients and surgeries, and seek out new and innovative treatments. This can all be done with the goals of minimizing neurologic deterioration, attaining stability and deformity correction, minimizing pain and maximizing function, and ultimately improving the lives of patients sustaining these injuries.

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Flexion-Distraction Injuries of the Thoracolumbar Spine

INTRODUCTION

Thoracolumbar injuries have been documented as early as the construction period of the great pyramids of Egypt (2600–2200 BC).[1–3] Since then, spinal injuries have accounted for more than 150,000 new hospitalizations per year in the United States.[4] The majority of such injuries occur in young adults and are largely attributed to high-energy traumatic events (i.e., motor vehicle accidents, high-level falls).[5–7] Based on a multicenter, 2-year prospective study reported by Gertzbein[8] in association with the Scoliosis Research Society, 52% of injuries occur between T11 and L1, 32% of injuries are noted between L1 and L5, and 16% of injuries manifest between T1 and T10. Although various forces account for spinal fractures, a preponderance of vertebral injuries result from distraction forces, which are commonly associated with damage to intra-abdominal viscera and vasculature.[9–16] As such, flexion-distraction injuries of the thoracolumbar spine are a serious concern that warrants careful evaluation and treatment.

EPIDEMIOLOGY

Flexion-distraction injuries represent up to 15% of all thoracolumbar trauma.[6-8,11,17,18] In the adult, such injuries occur primarily in males at the thoracolumbar junction (T11 to L2) and in the contiguous vertebrae or disk in up to 83% of patients.[6,7,17,19] These injuries occur, on average, during the second or third decades of life, but precise estimates cannot be ascertained because of sampling bias associated with many reports.[6,19,20] The majority of flexion-distraction injuries stem from motor vehicle accidents.[19] In addition, an increased risk of occurrence and degree of injury may be associated with the presence of altered bone metabolism, posture, event, and age.[21–23]

CLASSIFICATION

A flexion-distraction injury of the spine is commonly referred to as a Chance fracture. However, the classification of such an injury is a bit more elaborate. According to the literature, in 1948 Chance[24] briefly noted his experience with three patients who sustained "flexion fractures of the spine." Although the spinal level of the fracture in each patient was not reported, Chance described such fractures as posterior arch disruptions with a "horizontal splitting of the spine and neural arch, ending in an upward curve which usually reaches the upper surface of the body just in front of the neural foramen."[24] Almost 2 decades later, Howland et al.[25] reported their experience with a patient involved in a motor vehicle accident who sustained a similar injury as described by Chance. The patient was restrained with a lap seatbelt that acted as a fulcrum over which hyperflexion occurred as the body suddenly decelerated. As a result, the authors coined the term "Chance fracture," which has become synonymous with seatbelt-type injuries (Fig. 35-1). However, Bohler[26] noted that Heuritsch made drawings describing a Chance fracture 16 years earlier than Chance's publication. Nevertheless, shortly following the report by Howland and colleagues, Smith and Kaufer[15,27] further stressed the elements of hyperflexion and distraction forces at play in the development of a Chance fracture. The authors stated that injury was attributed to the patient in forward flexion and "submarining" under a high-riding lap seatbelt, which produced vertebral distraction and kept the pelvis and hips immobilized. The authors further proposed that minimal or no forward displacement of the vertebra is noted in flexion-distraction fractures and that such injuries may involve bone, soft tissue, or both.

Classification schemes of thoracolumbar instability based on column concepts or biomechanical models have been developed. Although various concepts have been proposed to address thoracolumbar injuries,[28–35] the Denis three-column model is widely used by clinicians today.[17,18,28–35] The Denis classification consists of an anterior column (anterior longitudinal ligament, anterior half of the vertebral body and disk), a middle column (posterior half of the vertebral body and disk, posterior longitudinal ligament), and a posterior column (pedicles, lamina, spinous processes, facets, ligaments) (Fig. 35-2). Biomechanical validity of such a model

Fig. 35-1 Mechanism of Chance fractures: Forward flexion over a high-riding lap-type seatbelt, producing vertebral distraction. (Adapted from Gumley G, Taylor TK, Ryan MD: Distraction fractures of the lumbar spine. J Bone Joint Surg Br 64:520–525, 1982.)

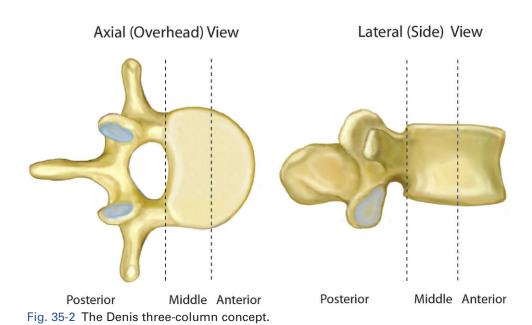has underlined the importance of the middle column in the mechanical stability of a thoracolumbar fracture.[36]

Flexion-distraction injuries of the thoracolumbar spine are typically classified as classic or ligamentous Chance fractures. In the classic Chance fracture, the anterior bending moment over the fulcrum and the associated tensile and shear forces acting on the spinal column create an elongation of the posterior column and disruption of the posterior and middle columns, with compression of the anterior column. However, the anterior column may also rupture on distraction when the fulcrum of rotation is anterior to the anterior column.[7,15,19,37] Ligamentous chance fractures involve disruption of the poste-

rior ligamentous structures extending through the anterior column via the disk space. Variants have been noted to occur and entail a combination of bone and soft tissue injury. Denis[17] classified four subtypes (A–D) of flexion-distraction injuries, based on the number of levels involved and if the injury occurred through bone or ligament (Fig. 35-3). Because the middle column is always involved in flexion-distraction injuries, classifications based on the occurrence of posterior and anterior located flexion-distraction fractures have also been proposed by Gumley et al.[11] and Gertzbein and Court-Brown,[7] respectively. Based on such classifications of posterior (type I–III) and anterior (type A, B, C1, C2) flexion-distraction fractures, Gertzbein and Court-Brown[7] noted that the posterior type II (fracture line at the interspinous process, lamina, pedicles, and entering the vertebral body; 65% of patients) and anterior Type C2 (disruption through the inferior endplate; 50% of patients) injuries are the most common. In addition, compression fractures and, to a lesser extent, burst fractures of the vertebral body have been noted to occur in flexion-distraction injuries, as a result of the high-velocity recoil following the primary fracture.[7,19]

CLINICAL PRESENTATION

In patients sustaining flexion-distraction injuries, evaluation of potential intraabdominal injuries should be well addressed. A large percentage of individuals sustaining flexion-distraction injuries sustain intraabdominal trauma involving viscera and vasculature structures.[9–16] Often, such injuries are missed and contribute to the morbidity of the patient.

The majority of flexion-distraction injuries of the thoracolumbar spine are purely bony fractures not associated with neurologic compromise. The exception occurs when a sig-
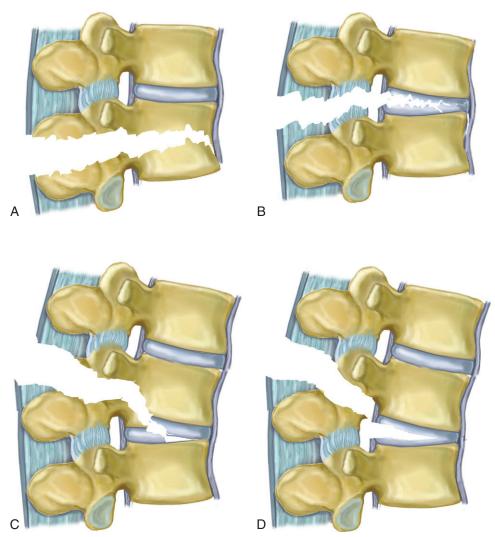
Axial (Overhead) View   Lateral (Side) View

Posterior   Middle Anterior   Posterior   Middle Anterior

Fig. 35-2 The Denis three-column concept.

Fig. 35-3 The Denis classification of flexion-distraction injuries. These may occur at one level through the bone *(A)*; at one level through the ligaments and disk *(B)*; at two levels, with the middle column injured through the bone *(C)*; or at two levels with the middle column injured through the ligament and disk *(D).*

nificant amount of translation is noted and the segment becomes more unstable. In settings of fracture-dislocations, the spinal column has complete failure of all three columns in tension, and intraabdominal injuries are, for the most part, present and entail rupture of a retroperitoneal organ. Paraplegia has been noted in patients with flexion-distraction injuries, fracture-dislocations[15] and in burst fractures.[7,37,38]

CLINICAL MANAGEMENT

DIAGNOSTIC TESTS

Following spinal injury, a complete neurologic evaluation should be performed to note sensory and motor function. The assessment of spinal instability is essential in determining treatment options and can be conducted radiographically to evaluate the integrity of both the osseous and ligamentous structures. In the event a patient is suspected to have a thoracolumbar injury, plain radiographs (anteroposterior and lateral views) of the entire spine should be obtained. Lateral plain radiographic evaluation can provide information of the spinal alignment and the integrity of the osseous elements. The treating physician should be cognizant of the various manifestations associated with spinal fractures. The lateral radiograph provides immediate assessment of any three-column disruption. Elongation of the posterior vertebral elements is evident on lateral and anteroposterior examination, whereby changes in the interspinous and/or interpedicular distances may be noted indicating posterior element injury. Fractures through the vertebral body and fracturedislocations can also be assessed via the lateral radiograph.

Computed tomographic (CT) imaging should be obtained in patients with flexion-distraction injuries to evaluate

the occurrence of intraabdominal injuries.[16] In addition, the presence of burst fractures are best visualized with CT imaging. Approximately 25% of burst fractures are misdiagnosed as compression fractures on plain radiographs.[39] In addition, CT imaging is useful in evaluating the integrity of all three columns.

Magnetic resonance imaging (MRI) is the modality of choice to evaluate for the presence of spinal cord injury, disk herniation, hematoma, edema, and ligamentous injury. MRI is ideal in determining the presence and extent of neural compromise and of ligamentous disruption in flexion-distraction injuries. Evaluation of the integrity of the ligamentous structures, primarily the posterior osteoligamentous structures, in flexion-distraction injuries is paramount and may dictate management options.

NONOPERATIVE APPROACHES

Management of an adult patient with a flexion-distraction thoracolumbar injury is largely dependent on the degree of bone and soft tissue involvement. In the young patient with a typical Chance fracture in which the injury is primarily osseous in nature, external immobilization in a hyperextension brace may lead to a good prognosis for healing.[40] In adults, because of many underlying systemic factors associated with age, lifestyle risk factors (i.e., obesity, long-term smoking), the existence of concomitant preexisting spine pathology (i.e., degeneration, deformity), and other comorbidities, management options are less straightforward. In the event of a purely osseous injury with no neurologic deficits, nonoperative means by bed rest, casting, bracing, or a combination of these may be highly effective.[11] Physiologic loads cannot be placed on the spinal column for up to 6 weeks to facilitate healing and maintain alignment.

INDICATIONS AND CONTRAINDICATIONS FOR SURGERY

The goals of surgery are to restore spinal alignment, hasten recovery, improve neurologic deficit, prevent secondary neural injury, and to reestablish spinal stability. According to biomechanical studies, thoracolumbar spinal segments will fail with weight bearing if the vertebral height loss and angulation are 50% and 20 degrees or more, respectively.[41,42] In the event of a soft tissue injury, nonoperative means to establish healing is not optimal because such an injury heals by scarring, is biomechanically inferior, and is riddled with acute and chronic instability; thus, surgery is recommended for soft tissue injuries. Furthermore, ligamentous injuries with significant translation necessitate surgical treatment. If spinal realignment and reduction cannot be adequately obtained via nonoperative treatment, surgery should be considered. Also, in light of undertaking nonoperative measures,

if neurologic deficit is progressing, malalignment is noted, and pain is increasing, surgical intervention should then be pursued.

OPERATIVE PROCEDURE AND TECHNIQUE

No consensus currently exists regarding the optimal surgical intervention in patients with flexion-distraction fractures. Some authors contend that multilevel instrumentation providing segmental fixation allowing for both compression and distraction should be performed.[7,43–45] Other surgeons contend that shorter constructs be used to preserve motion segments while affording adequate stability for healing.[6,20,46,47] However, the varied manifestations of flexion-distraction injuries documented in the literature stress that each case is individualized and that the treating physician must conduct a thorough physical and radiographic evaluation of the patient. Varying degrees of injury involving the bone, ligaments, disk, and neural elements, resulting in a range of spinal deformities and preexisting medical comorbidities in adults, may complicate treatment. Furthermore, surgical planning should address contiguous and noncontiguous injuries that may be present in addition to the primary site of injury.[19]

OPERATING ROOM SETUP/TECHNIQUE

The patient is placed in a prone position on a radiolucent operating room table (Jackson). The surgeon has the option of using supportive cushions under the chest and both iliac crests, which may aide in restoring sagittal contour via a postural reduction. Care is taken with patient positioning to keep the abdomen free, thereby decreasing venous hypertension and consequently decreasing venous bleeding. Furthermore, care should be taken to ensure that the anterior chest wall clears the operating table so that adequate chest expansion can occur during the surgical procedure. Fluoroscopy in both the lateral and anteroposterior plane can be used to assess the adequacy of reduction and for placement of pedicular instrumentation.

In the event that the injury involves ligamentous disruption at a single level, surgical intervention is sought to restore the posterior tension band.[20,48] Nonoperative treatment is contraindicated in primarily ligamentous injuries, which have a poor healing rate because of the avascular nature of such structures.[49] Therefore, in such cases, posterior, short-segment, pedicular-screw instrumentation and posterolateral fusions can achieve sufficient stability, preserve adjacent motion segments, and maintain alignment. If significant posterior ligamentous disruption is noted, excessive posterior distraction forces should be avoided to prevent catastrophic distraction of the cauda equina or spinal cord. In select cases in which patients are neurologically intact, distraction and ligamentotaxis can be performed to obtain indirect posterior decompression.

Combined flexion-distraction injuries and compression or burst fractures need to be identified to reduce morbidities and to institute appropriate treatment.[47] Surgery in such situations is warranted to prevent neurologic deterioration and progressive deformity and to re-establish spinal stability. Short-segment, posterior-only fixation for flexion-distraction injuries, associated with compression fractures of the anterior column, is more successful in the lower lumbar region because of its lordotic curvature. In general, a conservative rule of thumb is to instrument and fuse two levels above and below the site of fracture to prevent potential instrumentation failure and nonunion.[50] In a study by Sar and Bilen,[51] the authors noted no implant failures and no loss of correction in patients sustaining flexion-distraction injuries with concomitant compression fractures who were treated with posterior instrumentation and fusion. In the event of a concomitant burst fracture with a flexion-distraction injury, reconstruction of the anterior column with instrumentation coupled with compression and fixation of the posterior elements may provide immediate spinal stabilization, obtain desired restoration of sagittal alignment, and avoid progressive deformity.[51]

COMPLICATION AVOIDANCE

In the event an anterior column compression failure is noted accompanying a flexion-distraction injury, tension is applied operatively to the anterior longitudinal ligament with a cantilever hook or screw-rod construct. Caution should be used because overdistraction of the middle or posterior columns may occur, resulting in disastrous lengthening of the neural elements (Figs. 35-4, *A* and *B*). As a precautionary measure to avoid overdistraction, a temporary wire is secured around the posterior elements at the line of fracture while the anterior column is being distracted (Figs. 35-4, *C* and *D*). If the injury entails a relatively intact bony middle column, short, rigid posterior fixation one level above and below the site of injury provides sufficient compression and stability.[47,52] However, if the middle column is unstable, operative intervention
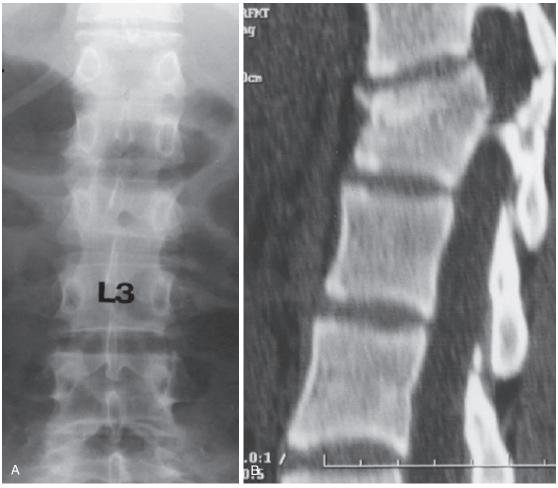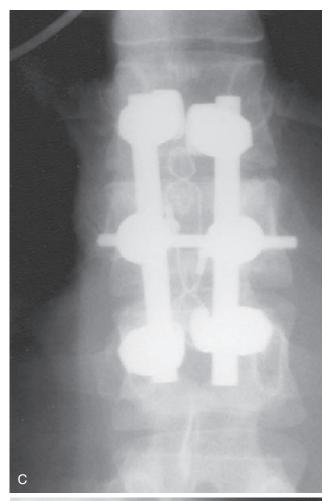

Fig. 35-4 A patient sustaining a flexion-distraction injury demonstrating on *(A)* AP plain radiograph and *(B)* lateral CT imaging disruption of the posterior ligamentous complex with a concomitant anterior vertebral body compression fracture. *C, D,* A short hook-rod construct placed in compression was used to restore the posterior tension band.
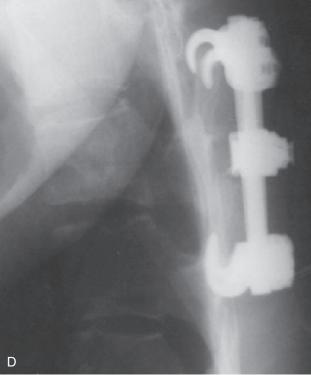
C

D

Fig. 35-4, cont'd *C, D*

applying compressive forces to reduce the fracture and reestablish the posterior tension band should proceed with caution to avoid the risk of retropulsion of bony or disk fragments into the spinal canal.[50,53,54]

SURGICAL OUTCOME

One of the most catastrophic complications following operative treatment of thoracolumbar burst fractures is neurologic deterioration. Such an event may be attributed to the extent of spinal cord injury, improper patient positioning, improper application of instrumentation, and intraoperative techniques to overdistract, overcompress, or reduce the fracture.[55] Nonunion, lack of fracture healing, and deformity progression following surgery may result in excessive biomechanical forces causing spinal instrumentation to fail. Pseudarthrosis rates are higher in posterior-only instrumentations and fusions because of the high tensile forces but may vary between bone graft substrates.[50,56–59] Patients undergoing an anterior lumbosacral exposure are at risk of potential damage to the autonomic nervous system, resulting in retrograde ejaculation in males in up to 8% of patients.[60–62] Such complications are directly related to the surgeon's experience with the anterior approach.[63] The development of deep spinal infections have been noted to occur in up to 10% of patients, requiring multiple irrigation and debridements.[49,50,64]

CONCLUSION

Flexion-distraction injuries of the thoracolumbar spine represent 15% of all thoracolumbar trauma. Such spinal injuries are a result of disruption of osseous and ligamentous spinal stabilizers, resulting from high tensile distraction forces with concomitant shear forces exerted in tension. Radiographic evaluation of such injuries should include assessment of spinal stability, extent of bone and ligamentous injury, intra-abdominal injury, and the risk of neurologic compromise. Radiographic evaluation should be multimodal and entail plain radiographs, CT imaging, and MRI. Management of such injuries in the adult are dependent on the extent and location of injury, age, underlying systemic factors, preexisting spinal pathology, and other risk factors that may impede fracture healing. The main goal of fracture management is to reestablish sagittal balance and spinal stability, while preserving neurologic function and preventing progression of deformity. As such, surgical intervention is often preferred in adults, especially in ligamentous injuries, because fracture healing is predictable, rehabilitation is expedited, and the use of external immobilization is minimized.

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Fracture-Dislocations of the Thoracolumbar Spine

INTRODUCTION

Fracture-dislocation injuries represent an uncommon but potentially devastating spine fracture. They account for 16% of spine fractures and are most common at the thoracolumbar junction.[1] The rate of neurologic deficit, however, is high when compared with other fractures. In the original Magerl et al.[2] article, these fractures had a 30% to 50% incidence of neurologic deficit, whereas 60% of patients in the Denis description had a neurologic deficit. Neurologic deficit associated with this type of fracture is a result of the high-energy force required to induce a fracture dislocation of the spine. In the mature spine, the thoracolumbar segment represents a transition point from the relatively rigid thoracic spine to a more mobile lumbar spine. Although this fracture is more common in adults, it also has been described in infants. However, fracture-dislocation injuries in infants are uncommon because of the elasticity of the immature spine. Given the involvement of all columns and the high rate of neurologic injury, treatment is primarily surgical. Nowadays, the preferred surgical approach is segmental fixation with pedicle screws.

CLASSIFICATION SYSTEMS

A number of classification systems exist for spine fractures. Holdsworth[3] described five categories of injury according to the mechanism of injury. Whitesides[4] subsequently defined the two-column concept of the spine, whereas Louis described three columns consisting of the vertebral bodies and the two articular pillars. Other systems include those described by Roy-Camille et al.,[5] McAfee et al.,[6] and White and Panjabi.[7] However, the two most recognized and used spine fracture classification systems are the Denis and the AO (as proposed by Magerl). According to the three-column Denis classification system, the four types of injuries are compression, burst, seat-belt, or fracture-dislocation.[1] The defining characteristic of fracture-dislocations is three-column failure associated with subluxation or dislocation. Uncommonly, the patient may present in a reduced position, but a careful radiographic analysis will reveal the tri-columnar fracture pattern.[1]

The mechanism in fracture-dislocation injuries involves rotation and shear combined with compression in the anterior column and distraction in the middle and posterior columns. The consequent failure of all three columns leads to dislocation and/or subluxation.[1]

Three subtypes of fracture-dislocation have been described within the Denis system (Table 36-1). These include the most common flexion-rotation (type A), shear (type B), and flexion-distraction (type C). Type A (84% of all fracture-dislocations) combine anterior wedging with disruption of ligamentous structures of the middle and posterior columns (Fig. 36-1). This leads to anterior wedging, but preserved posterior body height, and a fracture of the facet resulting from rotation. On the AP x-rays, the vertebral body is rotated compared with the adjacent level. The fracture may involve the disk or extend into the adjacent vertebral body.

Shear fractures (type B) are 10% of all fracture-dislocations and involve disruption of all three columns (including the anterior longitudinal ligament) from an anteroposterior (AP) or posteroanterior (PA, more common) directed force (Fig. 36-2). The vertebral body is intact, and the direction of displacement determines the AP or PA subtype.

The third subtype (flexion-distraction, type C) resembles a Chance fracture but includes the required subluxation that defines a fracture-dislocation injury (Fig. 36-3). They are the least common and occur in only 6% of cases. In Denis' original description, the anterior longitudinal ligament is maintained, but the other ligamentous structures (posterior longitudinal, disk space, interspinous) are disrupted. Unlike type A fractures, this subtype does not have a rotational component and, therefore, lacks facet fractures.

In 1994, Magerl et al.[2] described the comprehensive, but more complex AO fracture classification system. Three types of fractures were described. Defining characteristics of

TABLE 36-1   Denis Classification of Fracture-Dislocation Injuries

TYPE	MECHANISM	RADIOGRAPHIC FINDINGS
A	Flexion-rotation	Anterior vertebral wedging Rotation → facet fracture → May involve disk or body
B	Shear	Vertebral body intact (in both) PA–anterior dislocation: posterior arch fracture/ free floating lamina AP–posterior dislocation: no free plating lamina
C	Flexion-dislocation	No rotation (no facet fracture) ← unlike Type A Vertebral body offset Similar to Chance fracture but with dislocation

each of the three types include compression of the vertebral body in type A, two-column injury with distraction (as a result of tensile force) in type B, and two-column injury with rotation (as a result of axial torque) in type C. Each type is further divided into three subtypes with even further specifications in each subtype. Fracture-dislocation injuries with associated neurologic deficit can occur in either type B or type C fractures (Table 36-2). For instance, type B1 and B2 injuries are flexion-distraction fractures, whereas type B3 injuries result from hyperextension-shear forces with resultant subluxation. Type C1 injuries are compression injuries of the vertebral body with rotation, C2 are similar to B2 but with rotation, and C3 are rotational-shear injuries. Although the Denis and AO systems are widely recognized, interpretation issues cause a lack of widespread acceptance. For instance, a recent article describes the reliability and reproducibility among both the Denis and AO systems as moderate at best and indicates the need for a better system.[8]

FRACTURE-DISLOCATION INJURIES IN CHILDREN

The incidence of spine fractures in the pediatric subpopulation is 2% to 7%.[9–13] However, because of the relatively large size of the head in relation to the body, the majority of spine injuries in kids involve the cervical spine.[14] Consequently, there is relatively sparse literature available to guide the surgeon in the management of thoracolumbar shear injuries in children. In addition, the various spine fracture classification systems (e.g., Holdsworth,[3] McAfee et al.,[6] AO[2], Denis[1]) have not been validated in children and therefore are limited in application to kids. For instance, the youngest patient of the 412 injuries in the original Denis description was 17.

The presence of growth plates determines the spine fracture type in kids. The spine of a child differs from that of an adult in terms of the flexibility of the supporting structures,

growth plates, and elasticity and compressibility of the bone.[12,14,15] In the immature vertebrae, the cartilaginous end-plates that separate the adjacent disks gradually fuse to form an annular apophysis that is divided from the vertebral body by a thin layer of cartilage. Fracture-dislocation is more common in the adolescent and older child as opposed to infants. However, this fracture has been described in infants, specifically as a result of child abuse.[16–18] The presumed mechanism was a hyperflexion or hyperextension injury from the child abuse. However, fracture-dislocation injuries in infants are limited to case reports. Therefore, it is difficult to extrapolate the incidence and treatment guidelines to all children.

TREATMENT OPTIONS

NONOPERATIVE TREATMENT

In both the AO and Denis classification systems, fracture-dislocation injuries are considered unstable because of failure of all spinal columns. However, in children younger than 8 years, certain subtypes of these fractures can be managed by closed reduction and bracing.[19] A fracture through the disk or endplate/cartilaginous interface can appear as fracture dislocations on radiographic imaging, but a closer analysis will reveal that these injuries are periosteal tube fractures that often heal with closed reduction only.[19]

Before the advent of instrumentation in spinal surgery, most spine fractures were treated conservatively with closed reduction and bracing. Likewise, thoracolumbar fractures have been reported to be successfully treated nonoperatively.[20–22] However, the disadvantages associated with conservative therapy as compared with internal fixation include a higher rate of long-term deformity, longer hospitalization, delayed rehabilitation, and a higher systemic complication rate.[23–26] Consequently, advancements in surgical technique, a better understanding of the biomechanical properties of the spine, and superior instrumentation have replaced
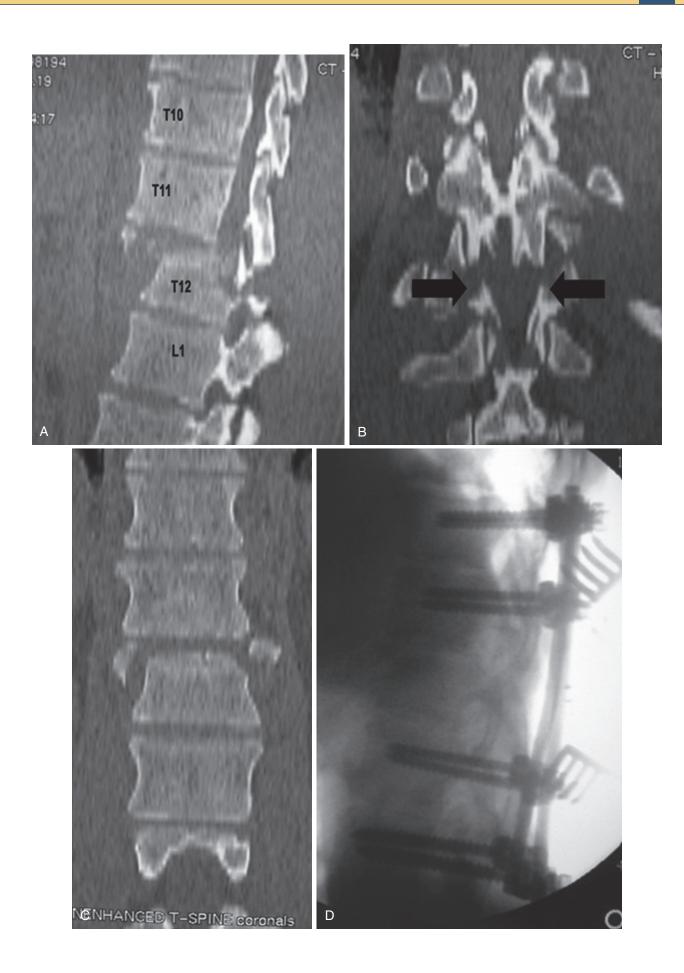
Fig. 36-1 Denis fracture-dislocation/flexion-rotation type (type A). *A,* Anterior dislocation with compression injury of T12 body. *B,* Note fracture through facets *(arrows),* which indicate rotational force. *C,* Dislocation can be through disk (as in this case) or body. *D,* Fracture treated with segmental pedicle screws two levels above and below with good reduction of fracture.
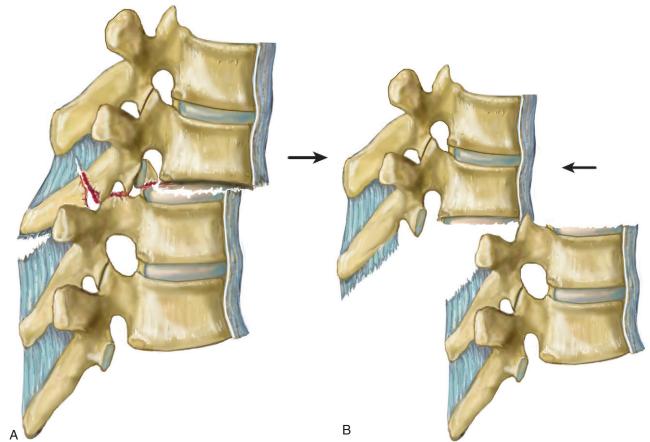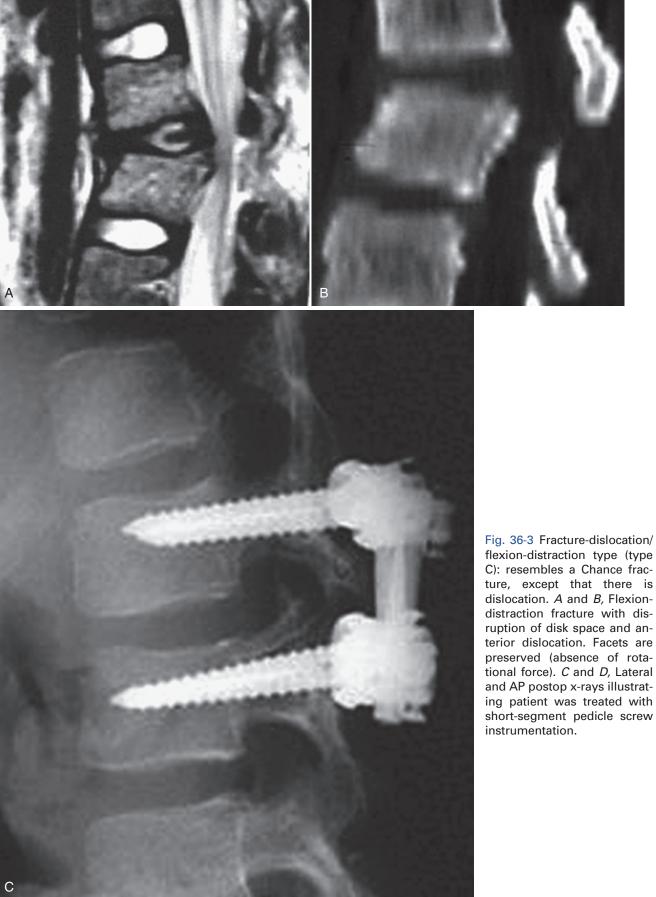

A

B

Fig. 36-2 Denis fracture-dislocation/shear type (type B): vertebral body is intact. *A,* PA type (most common): Posterior arch is fractured and can lead to "floating lamina." *B,* AP type (less common): No free floating lamina. Illustration adapted from Ref. 1.

conservative therapy as the treatment of choice in thoracolumbar fracture-dislocations.

OPERATIVE TREATMENT

Harrington rods are an early example of spinal fixation devices used for treatment of thoracolumbar fractures. Although the Harrington rod was traditionally used in corrective scoliosis surgery, Holdsworth popularized their use in thoracolumbar shear fractures.[27] Over the years, numerous authors have published their results using Harrington

rod instrumentation in treating fracture-dislocation injuries.[26,28,29] However, a disadvantage in using Harrington instrumentation is that two or more intact vertebrae above and below the injured segment are included to achieve stability.[30] Sasso and Cotler,[31] in a comparison of three fixation devices with Harrington rods for stabilization of fracture-dislocations in the thoracic spine, reported instrumenting an average of 6.5 levels using a Harrington rod/hook construct as compared with only three levels with pedicle screw fixation. In addition, despite the longer construct, McAfee et al.[32] demonstrated that the Harrington

Fig. 36-3 Fracture-dislocation/ flexion-distraction type (type C): resembles a Chance fracture, except that there is dislocation. *A* and *B,* Flexion-distraction fracture with disruption of disk space and anterior dislocation. Facets are preserved (absence of rotational force). *C* and *D,* Lateral and AP postop x-rays illustrating patient was treated with short-segment pedicle screw instrumentation.

Fig. 36-3, cont'd *D*

rods with or without segmental wiring were unable to resist rotation even at low torques. This is a significant disadvantage in treating an unstable rotationally induced three-column injury.

In addition to limiting the length of the construct, another important concept in spinal fixation surgery is preservation of sagittal balance. Overcorrection of the normal thoracolumbar spine curvature can result in a painful, and at times debilitating, condition known as "flat back syndrome" and can result in not only back pain but possibly neurologic deficits. This is a well-known sequelae of the distraction-inducing Harrington

TABLE **36-2**   AO Classification of Fracture-Dislocation Injuries

TYPE	B1	B2	B3	C1	C2	C3
Incidence of neurologic deficit (%)	30	33	50	53	60	50

rod instrumentation.[33,34] Numerous studies have shown a progressive increase in the kyphotic angle after instrumentation of fractures involving the thoracolumbar region and is especially prevalent with Harrington rod/hook constructs.[31,35–38] One reason for this sagittal collapse in fracture-dislocation injuries of the spine is the lack of sufficient strength of the injured spine to maintain a counterforce to keep the laminar/hook construct intact.[38,39] Both biomechanical and clinical experience have documented failure of the Harrington instrumentation in resisting the moment arm in flexion and extension.[39–41] During flexion and lateral bending the rods and hooks can dissociate because of the lack of rigid fixation of the hooks to the rods[38] while the hooks can fracture the lamina or cause facet dislocation in extension. Finally, the majority of postoperative deformity after Harrington rods used for thoracolumbar fractures has been attributed to the inability of the injured disk to resist compression. Therefore, any stand-alone posterior fixation will result in some degree of angulation in long-term follow-up.[35] Overall, the reported failure rate of the Harrington instrumentation in spinal stabilization is 7% to 10%.[42–45] Modifications to the Harrington system including placing compression hooks and segmental sublaminar wiring of the construct have been shown to improve some of the biomechanical disadvantages of this fixation device.[46–48]

Luque rod instrumentation also has been commonly used for spinal fractures. Unlike Harrington instrumentation, sublaminar wiring to the spine couples the two rods and provides superior rigidity to rotational forces.[32] Whereas biomechanical studies have shown that the failure of Harrington rods occurs at the bone-metal interface, stress of the Luque rod instrumentation results in transfer of the force away from the bone-metal interface to the spinal column above and below the construct.[32] However, one disadvantage of Luque rod fixation is telescoping of the rods within the wiring resulting in the relative inability to resist axial loading forces.[49] Similar to Harrington rod fixation, a Luque rod/sublaminar wire construct requires extension of the rods over several levels above and below the injured level.[31] Another advantage of segmental fixation devices over nonsegmental Harrington rods is the reduced need for postoperative bracing. However, some authors such as Nasca[50] report significant loss of lordosis without bracing.

Another segmental spinal fusion device used to stabilize thoracolumbar fractures is the Cotrel-Dubousset (CD) system.[51–53] This technique uses up- and down-going segmental laminar hooks connected to parallel rods. One advantage of the CD system compared with the Luque rod construct is a higher axial stability.[48,54,55] CD instrumentation has a similar fusion rate as Luque instrumentation; however, the likelihood of neurologic injury is higher with Luque instrumentation because of the sublaminar wiring technique.[50,55]

Segmental pedicle screw fixation of the fractured spine has become more popular. The advantages of pedicle screw fixation include three-column fixation, shorter length of construct, solid fixation in the absence of intact lamina, correction of the axial deformity by rotation of the rod, and in situ contouring of the rod to accommodate pedicle alignment.[56,57] In choosing a construct for stabilization of a thoracolumbar fracture one has to consider the long-term biomechanical alterations induced by the construct.[58] In the mature spine, the thoracolumbar junction (T10 to L2) represents a transitional point from a relatively immobile thoracic spine to a more flexible lumbar region. Thus, extension of a construct over several segments in the lumbar spine can result not only in loss of physiologic range of motion and resultant junctional pain but also degeneration of the adjacent level facets from alteration of lumbar mechanics.[59] In this respect, a short-segment pedicle screw instrumentation can be advantageous over hook/rod or wire/rod posterior fixation. However, a common criticism of all stand-alone posterior stabilization instrumentations is the potential loss of sagittal deformity correction. Similar to the previously mentioned internal fixation devices, pedicle screw instrumentation also has been reported to lose postoperative deformity correction.

Stand-alone pedicle screw fixation possibly can fracture or fail from bending as a result of the stress placed on the construct.[60,61] This is a result of the pedicle screws functioning as a load-bearing cantilever that ultimately results in collapse of the instrumented segment.[62–64] A supplementation to the pedicle screw construct that has been reported to counter the stress on the pedicle screws is a combination pedicle screw/offset hook/rostral laminar claw.[57,65–67]

Biomechanical testing of this construct has shown to increase stiffness and to thus reduce the bending movement on the screws. Despite the limitations of pedicle screws, they represent a significant advantage over nonsegmental (i.e., Harrington rods) and posterior column-only segmental (i.e., Luque rods) instrumentation systems.

The mean loss of kyphosis correction in thoracolumbar fractures treated with stand-alone posterior pedicle screw fusion has been reported to be between 3 and 12 degrees.[61,64,68]

Furthermore, the rate of pedicle screw bending, fracture, and pullout has been reported to be as high as 54%.[31,61,64,68–70] Gaines et al.[71] introduced a new classification system called "load sharing" to address the ability of the anterior and middle column of the injured level in supporting a posterior pedicle screw fixation construct. The classification is composed of three categories: comminution/involvement of the vertebrae, apposition of fragments, and deformity correction. Each category is assigned a point system of 1 to 3. This system was developed after a comprehensive retrospective review of Gaines' surgical management of patients with thoracolumbar fractures and

Comminution/Involvement

Little

<30% Comminution on sagittal plane section CT

More

30% - 60% Comminution

Gross

> 60% Comminution

Apposition of Fragments

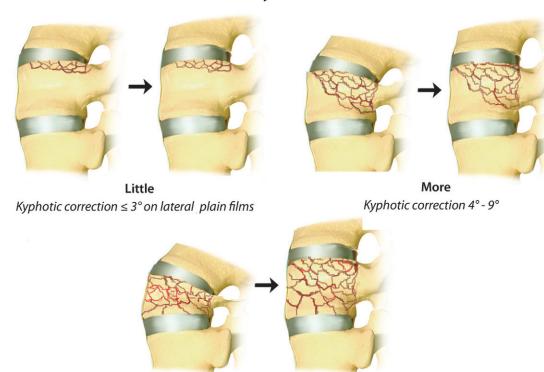**Minimal**

Minimal displacement on axial CT cut

Spread

At least 2 mm displacement of <50% cross section of body

Wide

At least 2 mm displacement of > 50% cross section of body

Deformity Correction

Little

Kyphotic correction ≤ 3° on lateral plain films

More

Kyphotic correction 4° - 9°

Most

Kyphotic correction ≥ 10°

Fig. 36-4 The load-sharing classification of spinal fractures.

experimental biomechanical studies.[72,73] Although this system was used as an algorithm for the treatment of burst fractures, the authors have successfully applied it to the management of thoracolumbar fracture-dislocations.[71] The authors report that all patients with a thoracolumbar fracture-dislocation and a score of 7 or higher on the load-sharing classification who were treated initially with a posterior short-segment pedicle screw fixation followed by anterior vertebrectomy and strut graft fusion had a smaller loss of correction compared with those who had a stand-alone posterior stabilization (Fig. 36-4).[71]

CONCLUSION

Fracture-dislocation injuries occur primarily in the adult and adolescent populations and have a high associated neurologic deficit. They are unstable injuries because of involvement of all columns and require surgical fixation. Surgical intervention is the treatment of choice with posterior pedicle screw fixation with or without supplemental laminar hooks. The majority of fracture-dislocations of the thoracolumbar spine can be treated with a stand-alone posterior pedicle screw fusion. Anterior/posterior fusion should be considered only if there is evidence that the anterior and middle column injury will result in postoperative kyphotic deformity.

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Low Lumbar Burst Fractures

INTRODUCTION

Burst or vertical compression fractures arise from the action of an axial force on a relatively straight spinal segment. Severe neurologic injuries, including both incomplete and complete spinal injuries, are associated with these fractures. It is thought that approximately 150,000 patients suffer spinal injuries in North America every year and that approximately 90% occur in the thoracolumbar region. In general, approximately 15% are considered burst fractures. Trauma, consisting mainly of vehicular accidents and falls, accounts for 75% of occurrences. It should be recognized that lower lumbar burst fractures account for only a small percentage of all spinal injuries.[1–4]

Burst fractures are produced by the delivery of an axial load on the neutral spine. Load sharing studies show that the vertebral bodies carry approximately two thirds of the axial force acting on the spine, with the posterior elements carrying up to one third when the spinal alignment is neutral; therefore, the anterior column (vertebral body and disk/annulus complex) absorbs the majority of the delivered forces. The vertebral bodies are composed of both a hard, brittle cortical layer and a spongy cancellous layer. The structural integrity of the vertebral body diminishes with time, and when people reach the age of 40 years, the trabecular bone content has already declined by approximately 30% compared with the maximum levels of youth. Systemic osteoporosis leads to a generalized weakness of the entire spinal column and becomes more common as the population ages.

The diameters of the lumbar vertebral bodies increase caudally. The vertebral canal diameter slowly increases from the lower thoracic spine to the lower lumbar spine. The proportion of canal space to neural tissue increases notably more from the thoracic spine to the sacrum, with spinal canal size increasing with tapering of the nerve roots.

The intervertebral disk is composed of an outer annulus and an inner nucleus. The outer annulus accounts for the majority of disk stiffness. The disk is weakest with tensile loading, which is how failure of this structure occurs. Disks are relatively immune to failure in compression, with the bone usually yielding first. When compressed, the disk stiffens and can act like a piston driving into the endplate, causing a vertebral body fracture.[5]

The anterior longitudinal ligament (ALL), supraspinous ligament, posterior longitudinal ligament (PLL), and ligamentum flavum all act to stabilize the spine. The main soft tissue determinants in spinal stability are the ALL, PLL, and disk. Ligaments fail by ripping and tearing under shear forces; compression does not lead to ligamentous failure.

The unique configuration of the neural elements at the thoracolumbar region accounts for the neurologic findings after injury. The spinal cord terminates as the conus medullaris at the L1-L2 level. Fractures at T10 can injure the spinal cord, those at T11-L2 can injure the conus, and those at or below L3 can affect the cauda equina. Conus medullaris and cauda equina lesions might have a better prognosis for functional recovery than thoracic spinal cord insults. Neural injuries usually occur in burst fractures when sudden forceful retropulsion of bone fragments into the cord, conus medullaris, or cauda equina occurs. Such an event initiates a molecular cascade, which can result in secondary insults. Among these events are vascular changes, inflammation, accumulation of neurotransmitters, metabolic derangements, electrolyte shifts, and edema formation. Distortion of the cord itself can cause dysfunction of the long tracts.

In terms of the origin of fracture initiation, the rigid thoracic spine directly opposes the more flexible lumbar spine. The net result is essentially a concentration of force centered on the thoracolumbar junction, most commonly at L1. The thoracic spine assumes kyphotic angulation with axial loading, whereas the lumbar spine becomes more lordotic. The thoracolumbar junction is subjective to a pure compressive load. The lower lumbar vertebrae, L3-L5, can withstand more net force before failure because of their larger bodies compared with L1 and L2, and the center of gravity lies closer to the lower vertebral bodies, resulting in fewer kyphotic fractures compared with the L1-L2 level. The relatively increased lordosis of the lower lumbar spine is somewhat protective in terms of burst fractures because a greater

amount of flexion force is required to produce a fracture than in the less lordotic upper lumbar spine.

A burst fracture is defined as a compressive-type injury resulting in failure of the vertebral body with outward circumferential displacements of bony fragments. Often, posterior retropulsion of a bony fragment into the spinal canal occurs, usually at the level of the pedicles (Fig. 37-1). Lower lumbar burst fractures can occur in conjunction with fractures of the posterior elements. Studies document high force concentrations at the base of the pedicle and posterosuperior part of the vertebral body near the pedicle, which explains why fractures occur at the level of the pedicles.[6,7]

Pain is the most common presenting symptom in patients with lower lumbar fractures. The discomfort usually is in the lumbar region, but compromise of an exiting nerve root or roots can produce radicular pain. Although the conus medullaris often is injured in conjunction with thoracolumbar injuries, this is uncommon with lower lumbar fractures. Significant central canal stenosis can result in partial or complete cauda equina syndrome. Radicular compression might produce not only pain but weakness and dermatomal sensory abnormalities. If the burst fracture is associated with other injuries, such as fractures of the transverse processes, a retroperitoneal hematoma can develop, which can produce a lumbosacral plexopathy.

The presentation of a trauma patient, particularly one with suspected spinal injury, can cause tension and anxiety for all concerned. Strict adherence to trauma protocols and their systematic approach ensures the greatest chance for clinical success. Maintenance of an adequate airway, ventilation, and

Fig. 37-1 Axial view computed tomographic scan of an L3 burst fracture shows retropulsion of bone and fractures at the level of the pedicle.

blood pressure are paramount. Once life-threatening injuries have been ruled out, a focused total spinal examination should be performed. This includes a careful and detailed neurologic evaluation in which all abnormalities are noted. Standardized validated scoring systems such as the American Spinal Injury Association scale can be helpful in following patients and speculating regarding prognosis. Abdominal bruising might suggest seatbelt injury. With the use of spinal precautions, the patient should be log rolled to the side, and the neck, back, and buttocks should be examined. All regions tender to palpation and percussion should be radiographically investigated.

CLINICAL MANAGEMENT

Plain films (radiographs), the mainstay of fracture diagnosis in decades past, are still important today at all levels in trauma situations. Anteroposterior and lateral view radiographs are helpful in quickly assessing alignment and surveying large spinal regions. Regarding burst fractures, vertebral body loss of height, disruption of the posterior vertebral body line, widening of the interpedicular distance, and enlargement of the disk interspaces might be noted (Fig. 37-2). Kyphosis, when present, is easily assessed, as are laminar fractures, which occasionally occur in association with burst fractures. Although visualization might be difficult, evidence of spinal canal compromise on plain films should be sought whenever loss of body height is involved. The use of plain films alone results in misdiagnosis 25% of the time, and one of five burst fractures cannot be differentiated from simple compression fractures by using this imaging modality.[8]

Historically, myelography has been used for the detection of compromise of the neural elements and dural tears and leaks resulting from burst fractures (Fig. 37-3). The incidence of dural tears in burst fracture studies is variable. Keenen et al.[9] reported a 7.7% rate. It is notable that 86% of the time, a neurologic deficit is associated with a dural defect.[10] Despite this significant correlation between dural tears and neurologic dysfunction, no direct correlation between dural violations and the degree of canal compromise has been shown.

Compared with plain radiography, computed tomography (CT) yields much more information regarding vertebral fractures and is the most sensitive test for determining fracture type. The sagittal reformatted images allow for the overall spinal alignment to be evaluated, and axial view images permit the exact degree of canal narrowing to be ascertained (Fig. 37-4). Fractures through the pedicles and vertebral body comminution can easily be seen on computed tomographic scans. Essentially, all elements of the fracture can be analyzed by CT. Measurements of the pedicles and vertebral bodies can aid in selecting spinal implant sizes when stabilization is required. When used in the trauma setting, some think that CT is less expensive, more sensitive, and faster than plain radiography for complete spine evaluation.[11]
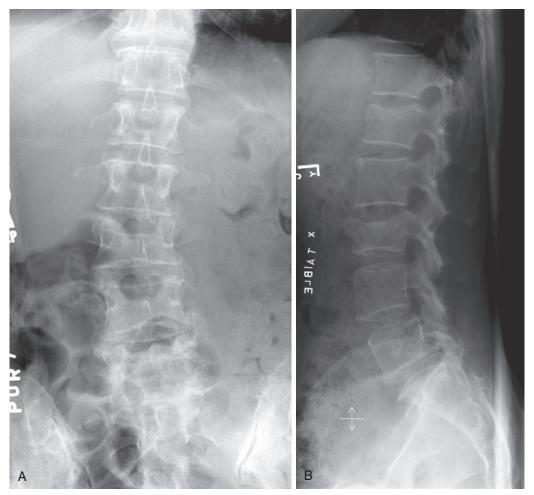
Fig. 37-2 Anteroposterior *(A)* and lateral *(B)* view radiographs show an L3 burst fracture. Note the widening of the pedicles at the level of injury.

Magnetic resonance imaging (MRI) should be considered as a test that complements but does not replace CT. MRI provides much greater resolution of soft tissue, the spinal cord, and disk material compared with CT (Fig. 37-5). MRI is an excellent modality for evaluation of entities such as disk injury, spinal cord contusion, nerve root entrapment, and spinal hematoma. MRI is specifically useful in identifying posterior element ligamentous disruption, with T1-weighted imaging showing higher specificity than T2-weighted imaging. The diagnostic accuracy of MRI in detecting disruptions in the supraspinous and interspinous ligaments is 90.5% and 94.3%, respectively.[12]

The ultimate goal of treatment is maximal recovery with the fewest side effects over a reasonable period of time. Proper spinal alignment should be restored or maintained, and the neural elements should be free of compression. Some patients clearly are candidates for either nonoperative therapy or surgery, but many are between these groups and the management decision-making process can thus be difficult. Neurologic deficits in the presence of compression, radiographic evidence of instability, and significant spinal deformity are key factors in determining the proper management.

A number of classification schemes have been proposed in an effort to guide treatment of injuries at the thoracolumbar junction. Although anatomic and biomechanical differences exist between these two regions, the classification systems can assist in the decision-making process for low lumbar burst fractures. Denis[2] conceptualized three columns as important in determining the structural integrity of the spine. Using this model, McAfee et al.[13] divided burst fractures into stable and unstable injuries. Stable burst fractures included those injuries that altered the integrity of the anterior and middle columns, whereas three-column injuries were deemed unstable. Although MRI was not used in the development of these schemes, it can provide a sensitive means of assessing the ligamentous integrity of the posterior column.[13]

The Spine Trauma Study Group (STSG) acknowledged that many biomechanical factors impact the management decision process for thoracolumbar injuries in terms of choosing the proper surgical therapy, but this group

Fig. 37-3 Anteroposterior *(A)* and lateral *(B)* view lumbar myelogram images of an L4 burst fracture show significant compression of the caudal equina.

ultimately identified three main injury characteristics as the most important: injury morphology, neurologic status, and the integrity of the posterior ligamentous complex.[14] By definition, a burst fracture involves injury to the anterior and middle columns. The importance placed by the STSG on posterior ligamentous injury as a major determinant for surgical intervention is consistent with other classification schemes.[13] According to the STSG, neurologically intact patients without posterior ligamentous instability do not need surgical stabilization. The STSG algorithm recommends that the neurologically intact patient with posterior ligamentous compromise be treated through a posterior approach. Patients with partial neurologic dysfunction and intact posterior ligaments should be treated with anterior decompression and instrumentation. If the local anatomy does not allow for such a procedure, the STSG recommends anterolateral decompression and posterior instrumentation. Incomplete neurologic deficit in conjunction with posterior ligament incompetence is managed with 360-degree decompression and stabilization. This group suggested that surgical intervention in neurologically complete patients be focused on achieving proper alignment and immediate stability.

Nonoperative management regimens include bed rest, spinal immobilization with a thoracolumbar orthosis, or a body cast. Potential complications with nonoperative therapy include the numerous problems that can develop with bed rest (pneumonia, deep venous thrombosis/pulmonary embolus, decubitus ulcer, etc.). Bracing can be associated with skin breakdown and loss of alignment. Surgery, however, is not without risk, and those patients who qualify for nonoperative management must fail this treatment before a surgical strategy is seriously considered.

OPERATIVE PROCEDURES AND TECHNIQUES

ANTERIOR APPROACHES

Anterior spinal surgery can optimize neural decompression and provide stable internal fixation across the spinal segments adjacent to the injury. For this disease entity, the major approach to the vertebral body is anterolateral. Such an exposure will allow for safe decompression of the spinal canal and reconstruction of the anterior spinal column. Ideally, patients

Fig. 37-4 L3 burst fracture. *A,* Axial view. *B,* Coronal reconstruction. *C,* Sagittal reconstruction.
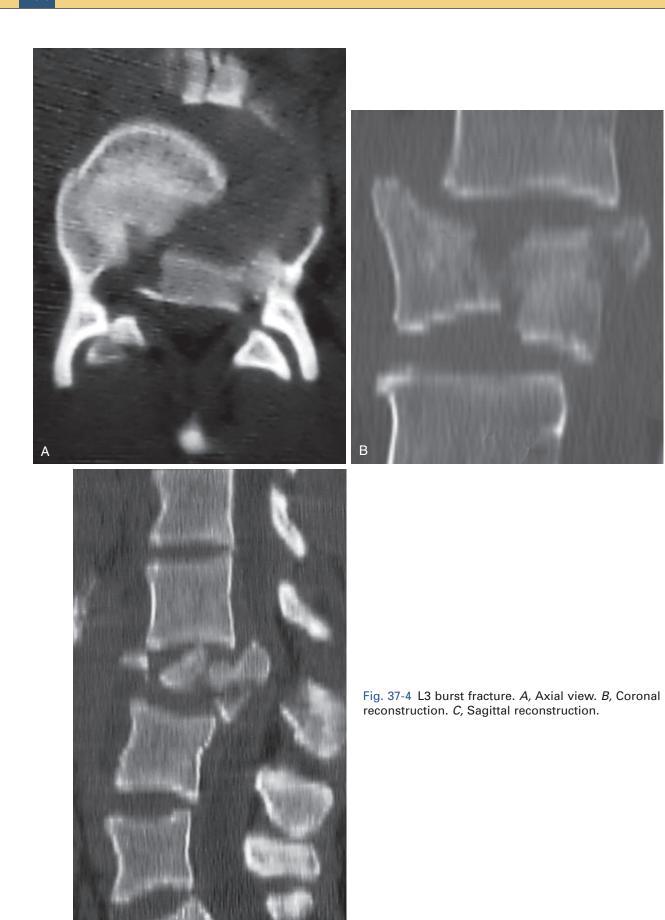
Fig. 37-5 Sagittal view T2-weighted magnetic resonance image of an L2 burst fracture.

are treated with a single-level decompressive corpectomy and reduction of any sagittal abnormality.

The lateral extracavitary approach is ideally suited for the lower lumbar region because it provides direct vision of the spinal canal without the risks of a laparotomy.[15,16] For this approach, the patient is placed in the supine position and a midline incision centered over the fracture is made. The incision curves laterally in the caudal region like a hockey stick. After subperiosteal dissection, the soft tissues are elevated and retracted medially. The iliac crest might hinder the exposure in the low lumbar regions. In these situations, it can be resected and used for graft substrate. The neurovascular elements are identified, and the dissection proceeds medially to the neural foramen. The pedicle above the foramen is removed, and the disks on either side of the injured vertebral body are identified and removed. The fractured vertebral body is removed and the thecal sac decompressed under direct vision without significant retraction of the neural elements. At this point, anterior and, if necessary, posterior instrumentation is placed.

The retroperitoneal approach offers a different route to this region. The transversalis fascia is exposed, which allows the surgeon to identify the peritoneum, and a dissection plane is developed just superficial to this structure. The peritoneum is retracted medially and dissected away from the psoas muscle. It is critical to avoid injury to a number of important structures when using this approach, including the genitofemoral nerve, iliac vessels, ureter, gonadal vein, sympathetic chain, and aorta.[17] The psoas muscle is ultimately dissected from the vertebral bodies, the disks removed above and below the injured segment, and decompression with reconstruction performed as mentioned previously. A separate incision might be necessary to place posterior instrumentation when decompressing with a retroperitoneal approach.

Proper reconstruction calls for an appropriate anterior load-bearing member combined with internal fixation across the injured segment. These goals can be accomplished with structural iliac autograft or allograft or a cage device (titanium, carbon, or polyetheretherketone) packed with autograft. Intraoperative radiographic assessment is essential to avoid overdistraction. The segments are further stabilized with anterior instrumentation. Bicortical fixation and appropriate compression to maximize load sharing should be achieved in all constructs (Fig. 37-6). Postoperative immobilization with an appropriate orthosis is necessary. When indicated, this approach is beneficial in that it allows for direct anterior decompression without neural retraction and anterior column reconstruction over a minimal number of levels.[18]

POSTERIOR APPROACHES

When indicated, posterior approaches provide a direct and relatively simple means of achieving internal fixation with an interbody and/or posterolateral fusion. The posterior approach can be performed in a minimally invasive manner, but this technique should be reserved for surgeons with experience in minimal access spinal surgery for degenerative disease. Significant anterior spinal canal compromise is an indication for an anterior approach. Moderate or minor canal compromise can be addressed through a posterior route if one subscribes to the notion of ligamentotaxis. The ligamentotaxis or annulotaxis theory is founded on the assumption that the posterior vertebral ligamentous complex will reduce fragments that are extruded into the canal if adequate distraction is achieved.[19–21]

Posterior stabilization is accomplished with pedicle screw fixation, and often, posterolateral fusion augments the instrumentation. If anterior decompression has been performed, the number of instrumented posterior segments should match the levels plated anteriorly. Ideally, a minimal number of levels are fused, but the exact definition of short-segment posterior fixation is controversial. Typically, this refers to fixation one level above and one level below the
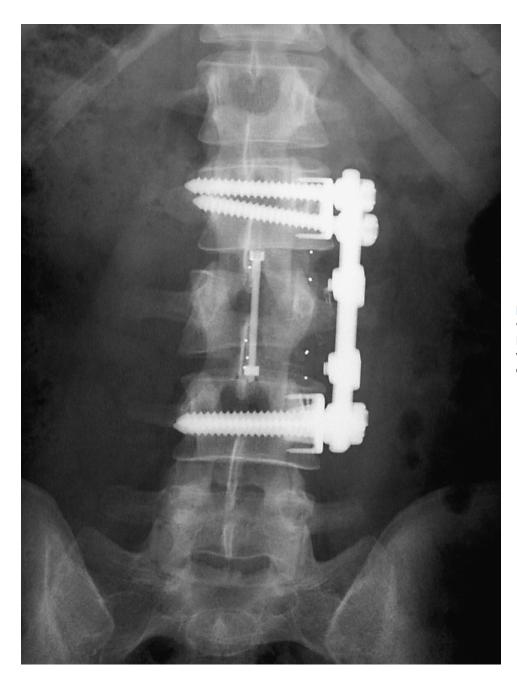
Fig. 37-6 Anteroposterior view radiograph of an L3 burst fracture reconstructed with stackable carbon fiber cages and a Kaneda plate.

fracture (two motion segments). Failure rates of such constructs when pedicle fixation alone is used can approach 50%; three-level short-segment fixation has therefore been described.[22,23] The strength of fixation is greatly increased by adding laminar hooks two levels above and one level below, which increases the length of the construct by only a single motion segment.[24,25]

Pedicle screw fixation is a common procedure, and the technical aspects should be well understood by surgeons performing spinal surgery. Care should be taken to carefully position patients who have burst fractures, and fluoroscopy is useful in maximizing proper alignment before the procedure

begins. Some degree of distraction usually is performed after the pedicle screws are placed. Distraction can help reduce fragments via ligamentotaxis, or the surgeon might choose to tamp fragments anteriorly using specially designed instruments or downbiting curettes. Distraction can promote kyphosis, so care should be taken to properly contour the rods to maintain lordosis. The longitudinal members should be cross-linked to maximize stability. A posterolateral fusion is performed in conjunction with the stabilization using autograft.

The complications of pedicle fixation include nerve root injury and the other usual potential problems, such as

infection, nonunion, and so forth. Posterior instrumentation alone can correct kyphosis but is not as effective in maintaining that correction compared with an anterior procedure.[18] Maintenance of sagittal balance is important, but currently, no solid evidence exists to indicate that the loss of lordosis, which might be associated with posterior instrumentation, significantly impacts patient outcomes.

SUMMARY

Low lumbar burst fractures are relatively uncommon spinal injuries. These injuries can be managed nonoperatively with anterior, posterior, or combined surgery, depending on a number of clinical and radiographic factors. The presence of neurologic deficit and posterior instability is critical in determining the need for operative care and the ideal management strategy. Despite all the thoughtful and appropriate work that has been performed in an effort to maximize outcomes in this group of patients, it is interesting to note that the greatest predictive factor of postoperative vitality might be the patient's mental health.[26]

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C H A P T E R

38

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Sacral Fractures

INTRODUCTION

The sacrum has been called by some the "keystone" of the pelvis. Some key functions of the sacrum include connecting the spine to the pelvis, and containment and protection of the neural elements that impart function to the bowel, bladder, sexual organs, and lower extremities. Sacral injuries can vary greatly in energy and mechanism. Treating sacral injuries requires a thorough knowledge of the three-dimensional anatomy of the sacrum, including the neural and supporting structures intimately associated with it. Treating acute sacral injuries often requires a team approach, including a spine surgeon and a trauma surgeon. Rehabilitation medicine often is involved later on to assist patients with functional recovery and adaptation.

ANATOMY

The sacrum is the most caudal structural segment of the spine. Its five vertebrae decrease in size, progressing in the caudal direction.[1] The five vertebral segments begin to fuse at approximately 15 years of age, and the fusion moves in a cephalad direction, completing at approximately age 25 years. The sacrum is tilted forward approximately 45 to 60 degrees in most patients, but inclinations as high as 90 degrees or as low as 10 degrees have been described.[2,3] In relation to the rest of the spine, a plumb line from C7 should pass through the posterior third of the S1 body. Deviation from this, especially anteriorly, has been implicated as a cause of back pain.[2] Some variability exists at the lumbosacral segment and can take the form of a sacralized L5, a lumbarized S1, congenital L5 to S1 fusion, or lack of fusion of S1 to S2.[1] It is important to recognize such dysmorphisms of the sacrum during preoperative planning to avoid problems with surgical procedures, because spinopelvic relationships, landmarks, and position of neurovascular structures might be distorted.[4]

Distribution of bone density is highly variable in the sacrum. The area of the sacral promontory contains the densest cancellous bone, with the bone in the ala decreasing in density. In some adults, alar areas that entirely lack cancellous

bone can be found (Fig. 38-1).[5,6] One implication of this heterogeneous bone distribution is the formation of a stress riser that can lead to fractures.

Dorsally, the sacrum follows a similar pattern as in the rest of the spine, but its posterior structures are fused. Spinous processes become the central ridge, and facet joints and transverse processes become the paracentral and lateral ridges, respectively. The pedicles are fused between sacral segments, forming the lateral borders of the neural canal. At the caudal sacrum, a hiatus exists where the posterior elements are not fused. The risk of fracture in the caudal sacrum increases with the increasing size of the sacral hiatus.[7]

Considering its location between the pelvis and lumbar spine, the sacrum performs a major role in lumbopelvic stability. Looking at an outlet view of the pelvis, one can see how the sacrum resembles the keystone of an archway providing inherent stability based on its geometry (Fig. 38-2).[8] Conversely, on an inlet view, the sacrum is arranged in what can be called a "reverse keystone," and stability in this plane depends on ligaments and other soft tissue structures. The ligamentous structures primarily responsible for sacropelvic stability are the interosseous ligaments and posterior and anterior sacroiliac ligaments. Of the sacroiliac ligaments, the posterior ligaments are far more robust, with the anterior ones contributing much less to overall stability. The sacrospinous and sacrotuberous ligaments also contribute to overall stability within the sacropelvic unit.[9] Ligamentous stability between the sacropelvic unit and the rest of the spine is provided, mainly by the iliolumbar ligaments from the L5 transverse process to the iliac crest and the sacrolumbar ligaments from the L5 transverse process to the anterosuperior sacrum and sacroiliac joint.[1]

The anatomy of the neural elements changes within the sacrum with the termination of the dural sac at approximately the S2 level. The filum terminale continues on to tether the tip of the dural sac to the coccyx. The sacral nerve roots move laterally as they near their exiting foramen. The dorsal roots supply sensory fibers to the skin and motor fibers to the paraspinous musculature. The ventral roots combine with the L4 and L5 roots to form the sacral plexus, which then gives off branches to the sciatic, pudendal, superior gluteal, and inferior gluteal nerves.[10] In addition, the ventral roots contribute to the sympathetic chain providing
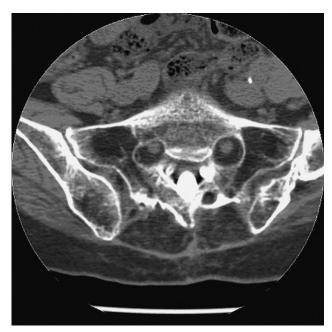
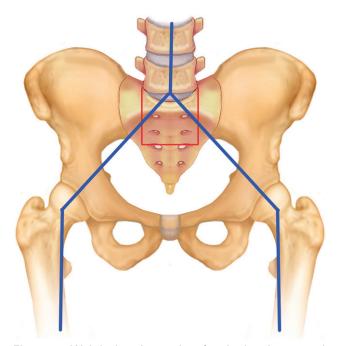Fig. 38-1 Bilateral sacral voids in a 75-year-old female patient.

Fig. 38-2 Weight-bearing axis of spinal column and pelvic ring.

bowel, bladder, and sexual function.[11] The diameter of the exiting sacral roots decreases precipitously with each successive root. In contrast, the diameter of each neural foramen does not decrease as rapidly. The clinical significance of this is that the upper sacral roots have less free space, which can predispose them to injury.[12]

The vascular anatomy of the sacrum can be variable, but some generalizations can be made. The middle sacral artery leaves the aortic bifurcation and descends in the midline over the anterior sacral promontory. The superior lateral sacral artery, a branch of the internal iliac artery, courses along the superior aspect of the sacroiliac joint to the lateral border of the S1-S2 foramina, where it gives off branches into the foramina. The inferior lateral sacral artery also comes from the internal iliac artery and courses along the caudal aspect of the sacroiliac joint, eventually anastomosing with the middle sacral artery. The lateral sacral artery also sends branches that pass through the ventral sacral foramina into the spinal canal. The venous system stems from the internal iliac veins and forms a presacral plexus with many anastomoses, including some to the epidural veins.[1,13]

Some other anatomic relationships have clinical significance for those treating sacral injuries and for those who perform surgery on or near the sacrum. One is the relationship of the rectum to the ventral sacrum. Below S3, the rectum lies immediately ventral to the sacrum. This makes it necessary to rule out injuries that communicate with the rectum.[13] Another important relationship is that of the L5 nerve root, which is draped over the sacral ala. This proximity makes the correct placement of screws in the area paramount, as a few millimeters of error could injure the nerve.[14]

CLASSIFICATION

In 1945, Bonnin[15] classified sacral fractures into those caused directly via impact to the sacrum from a blow or a projectile and those caused indirectly via forces transmitted through the pelvic ring. He showed that indirect injuries had more impact on spinopelvic stability and that injuries to the neural foramina were associated with a higher incidence of neurologic deficits, whereas fractures caused by direct mechanism tended to have very little effect on spinopelvic stability. Huittinen[16] classified sacral injuries based on the type of nerve injury in postmortem specimens. He divided injuries into traction, compression, and ruptures, of which only compression injuries were thought to be amenable to surgical management. Schmidek et al.[8] saw the pelvis and sacrum as a single functional unit and grouped sacral fractures with associated pelvic injuries. Sabiston and Wing[17] classified sacral fractures into those associated with pelvic injury, those in the lower segment alone, and those in the upper segment alone, finding increasing incidence of neurologic deficits in the latter groups. They went on to recommend conservative treatment for all types because they observed that the neurologic injury generally resolved. Other authors have described various sacral fractures with or without associated lumbosacral instability.[18,19]

One of the most widely used classification systems today is that presented by Denis et al.[12] based on location of the fracture line. Denis et al. divided the sacrum into zones I, II, and III,

with zone I fractures being lateral to the foramina, zone II fractures involving the neural foramina, and zone III fractures being medial to the foramina into the spinal canal (Fig. 38-3). In his series of 236 fractures, Denis et al. found a correlation between the zone of fracture and the likelihood of neurologic injury. Among zone I injuries, 5.9% had neurologic injury. This increased to 28.4% among zone II injuries, and in zone III injuries, the percentage of patients with neurologic injury was as high as 56.7%. Of those with zone III injuries, 76% had impairment of bowel, bladder, and sexual function.[12] Inspired by the work done by Denis et al., Gibbons et al.[20] later formulated a classification based on functional neurologic injury. Their classification divided fractures into the following four groups: (1) those with no neurologic sequelae, (2) those with injury causing sensory loss, (3) those with injury causing motor loss with or without sensory loss, and (4) those with injury causing bowel and bladder dysfunction.

INJURY PATTERNS

The incidence of neurologic injury is highest in Denis zone III fractures, but all zone III fractures are not equal. In 1979, Wiesel et al.[21] reported a case in which a patient with a longitudinal zone III sacral fracture was treated conservatively and had no neurologic compromise. They thought that the nerve roots were spared because they were displaced laterally as a result of the longitudinal fracture orientation. Others have presented series of patients with zone III injuries and lower incidences of neurologic compromise than would be expected. Hatem and West[22] reported a case of a zone III injury into the spinal canal with no neurologic
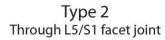

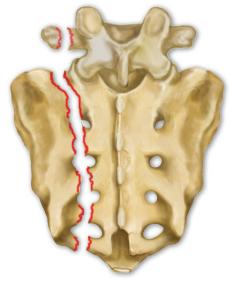Fig. 38-3 Denis classification of sacral fractures.

sequelae and, based on a review of the literature, suggested that sagittal injuries seem less likely to have associated neurologic deficits than other types of zone III injuries. Bellabarba et al.[23] presented the reports of 10 patients with sagittal zone III injuries; none had motor or sensory deficits but three had some form of sexual dysfunction. They thought that the fractures might represent a variant of pelvic anterior-posterior compression injury. It seems that longitudinal zone III fractures carry less risk of neurologic injury; however, Isler[24] suggested that the injuries might cause significant damage to the lumbosacral junction, making it unstable. He found that if the fracture line crossed through or medial to the S1 articular facet—a zone III injury—a 38% incidence of lumbosacral injury was present compared with 6% when the fracture line was lateral to the S1 facet (Fig. 38-4).
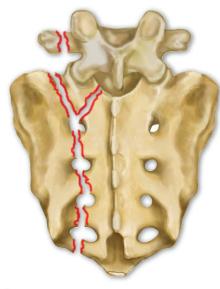
Although most sacral fractures occur in the sagittal plane, as early as 1969, Purser[25] described a fracture in the transverse plane through the upper sacrum and into both sacroiliac joints. Although Purser's report described a patient without significant neurologic injury, later reports of upper sacral transverse fractures have described a high rate of neurologic deficits in these patients.[26] It was postulated that these fractures might be analogous to traumatic spondylolisthesis of the lumbosacral junction, with the mechanism of injury being a flexion force across the upper sacrum that fails because of relative weakness of this segment.[8,18,19] Roy-Camille et al.[27] described and classified transverse upper sacral fractures or "suicidal jumper's fractures" into three types. Type 1 injuries were thought to be caused by simple bending. Type 2 injuries combined bending with subsequent posterior translation of the upper segment on the lower. Type 3 injuries were extension fractures with anterior translation of the upper segment on the lower. A fourth type was later added, which was described as a direct impact without flexion or extension (Fig. 38-5).[28] Transverse fractures of the lower sacral segments have also been reported in the literature. The mechanism of injury in these fractures is thought to be a direct blow to the coccyx and/or lower sacral segment that causes a flexion movement across the segment.[29] Based on the reports in the literature,[17,19,30–38] it is apparent that transverse fractures of both the upper and lower sacrum have a high likelihood of associated neurologic injury. Most of the early reports discuss transverse sacral fractures as though they are simple fractures; however, with the advent of newer radiographic techniques such as computed tomography (CT), we have observed that these fractures are almost always complex in nature. Many have classified them based on the shapes of their fracture lines: H, T, U, and λ.

Insufficiency fractures of the sacrum are extremely common, but in contrast to high-energy fractures, they carry a low risk of neurologic compromise.[39,40] These fractures often result from minor falls or can be the consequence of normal ambulation or gentle movement. Often, the patient has risk factors for osteoporosis, such as inactivity, advanced age, chronic corticosteroid use, malignancy, or menopause.

Type 1
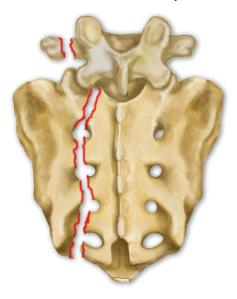Lateral to L5/S1 facet joint

Type 2
Through L5/S1 facet joint

Type 3
Medial to L5/S1 facet joint

Fig. 38-4 Isler classification of sacral fractures.

Because of this, it is important to screen for other fractures in common sites such as the vertebrae, proximal femora, and pelvis. Another contributing factor might be previous spinal fusion creating a long lever arm, which increases the stresses at the sacrum.[41–44] Lumbar scoliosis also has been identified as a factor because insufficiency fractures have been identified on the concave side of the curve. It is important to consider insufficiency fractures in the elderly patient who presents with low back pain because this has been shown to be a common complaint.[40] The generally benign nature of these fractures should not preclude a thorough neurologic evaluation

and close follow-up of the patient. A literature review by Jaquot et al.[45] showed a 2% incidence of neurologic injury in 493 cases. They reported that the true incidence is likely higher, however, because symptoms of bowel, bladder, and sexual function often are attributed to other causes in the elderly population when, in reality, they might be related to sacral injury.

Sacral stress fractures are far less common than insufficiency fractures. Sacral stress fractures occur in patients whose activity levels exceed the reparative process of bone, such as highly competitive athletes and military recruits. Generally,
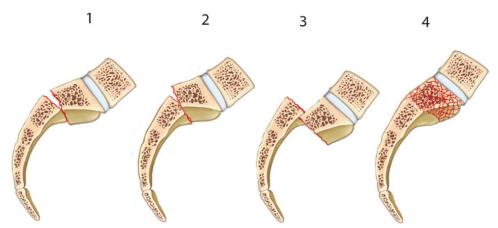
Fig. 38-5 Roy-Camille classification of sacral fractures with Strange Vognsen addition.

the bone is healthy, but in some cases, such as the case of a female athlete with amenorrhea, the fracture might be a combined stress and insufficiency fracture because such patients generally are osteopenic even though their activity levels imply otherwise. Stress fractures often have the same vague symptoms as insufficiency fractures, such as generalized low back pain.(Fig. 38-6).

EVALUATION

Whether caused by high- or low-energy mechanisms, sacral fractures have a high likelihood of being missed during initial evaluation. The series presented by Denis showed that half of the sacral fractures in patients who were neurologically intact were initially missed. If neurologic deficit was present, one third were missed.[12] Laasonen[46] thinks the reason sacral injuries are difficult to diagnose radiographically is the curved shape of the sacrum and the use of plain radiographs for screening. This being the case, a high index of suspicion is warranted in patients with complaints of low back pain and/ or a mechanism for sacral injury.

High-energy sacral fractures generally present as acute trauma to an emergency department. Patients with these fractures often have multiple injuries and require expeditious resuscitation and evaluation. Advanced Trauma Life Support (ATLS) protocols have been developed to assist in the acute resuscitation of these patients, first addressing the life-threatening injuries and cardiopulmonary status.[47] Once the patient has been stabilized, a secondary survey is conducted to identify any other injuries. The secondary survey includes a thorough examination of the spine and pelvis. The spinal examination starts with inspection and palpation for discoloration, step-offs, tender areas, ballotable areas, crepitus, and open wounds. The examination includes rectal and vaginal examinations to detect open fractures involving the rectum or vaginal vault, respectively. The presence of a large degloving injury has implications that might influence the treatment plan, as the risk of infection with surgical

intervention is significantly elevated.[48] The examination should also check for gross pelvic stability. If hemodynamic instability is present with an injury that enlarges the pelvic volume, the use of a sheet, clamp, or external fixator can decrease the pelvic volume and restore hemodynamic stability. If hemodynamic instability persists, workup for vascular injury ensues with angiographic evaluation. When a vascular injury is identified, it can sometimes be treated with immediate embolization or might need repair by a vascular surgeon.[49]

The neurologic examination is an important part of the survey once hemodynamic stability has been achieved. The examination includes a complete rectal examination for tone, voluntary control, and perianal pin sensation. In sacral injuries, the perianal sensory examination is possibly the most useful because it can detect injury to the highest number of caudal nerve roots. Conversely, the motor examination is somewhat limited in that it cannot evaluate roots below the S1 level. Nerve tension signs, such as a positive straight leg raise test, might be present, indicating that impingement of the lumbosacral plexus has occurred.[50–52] The neurologic examination should be repeated periodically because some causes of deficits, such as epidural hematoma, can present in a delayed fashion.[15] Electrodiagnostic studies also can be useful in the evaluation of sacral injury, especially in the obtunded patient.[8] Pudendal somatosensory-evoked potentials, combined with electromyography of the anal sphincter, allow detection of injury in the lower sacral roots.[53–56]

Conducting imaging studies is another important step in the diagnosis and management of sacral fractures. The anteroposterior view radiograph of the pelvis is the starting point in the trauma workup, but it can be difficult to diagnose many sacral injuries based solely on this view because of the geometry of the sacrum noted earlier in the chapter. Some radiographic indicators of sacral injury that require further workup include a paradoxical inlet view in which the sacrum is seen as an inlet view but the pelvis is seen in the anteroposterior view, abnormality in the sacral foramina, and disruption of the arcuate
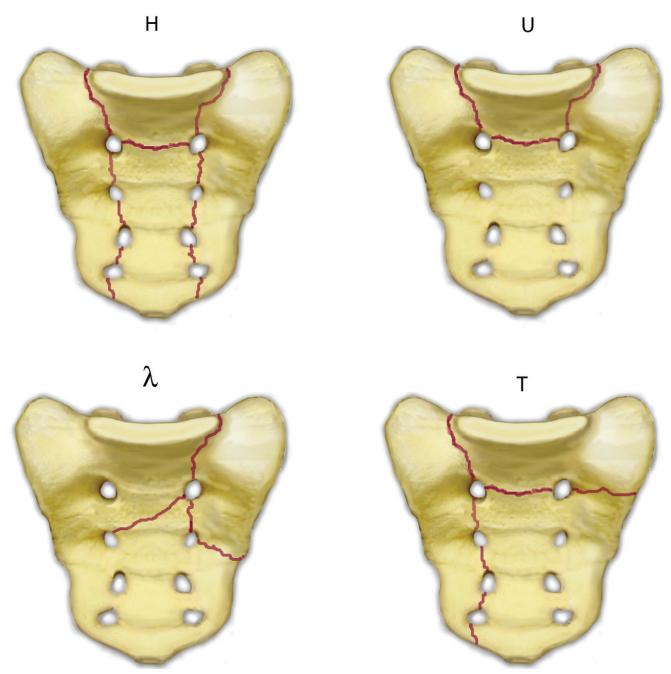
H

U

λ

T

Fig. 38-6 Roy-Camille classification of sacral fractures with Strange-Vognsen addition.

line. Other radiographic views that can help the treating physician diagnose injury include pelvic inlet and outlet views, true anteroposterior sacral view, and the lateral sacral view. CT is invaluable in the diagnosis and treatment of sacral injuries. CT allows detailed evaluation of the fracture pattern, provides insight into the mechanism of injury, and facilitates evaluation of the neural canal and neural foramina. CT also is an integral part of the general trauma workup, as it is used to evaluate the abdomen and chest for injury. Bone scans can be useful in assisting with the diagnosis of sacral stress or insuf-

ficiency fractures.[57] Magnetic resonance imaging also has some usefulness in diagnosing occult stress or insufficiency fractures[58] but has not been as useful in the initial evaluation of sacral fractures from high-energy mechanisms.

TREATMENT PRINCIPLES

Multiple factors must be considered when treating sacral fractures. The patient's overall medical condition must be optimized no matter what form of treatment is administered.

Concomitant injuries also must be addressed and might influence the type of treatment recommended. Benefits of early surgical stabilization of fractures have been shown in trauma patients with pulmonary injuries, because these patients are then able to mobilize earlier, thus decreasing the incidence of acute respiratory distress syndrome.[59,60] In patients with intracranial trauma, controversy still exists regarding the timing of surgical stabilization. Some have found a higher incidence of secondary brain injury in patients who were stabilized early, whereas others have not found that to be the case.[61,62] Once medical issues and other injuries have been evaluated, the decision regarding how to treat these injuries is based on the fracture pattern with consideration of the neurologic status.

Fracture displacement can be an indicator of stability of sacral fractures. Wide displacement generally infers more soft tissue injury, and the likelihood of further displacement is high. Fracture comminution is another means of inferring the energy absorbed at injury, and increasing comminution generally means less inherent stability. It might also mean less ability to use internal fixation because of the inability of screws to obtain purchase. One must be sure to consider the mechanism of injury regardless of the displacement or fracture comminution, because some fractures resulting from high-energy trauma might not present in a very displaced position but can still be unstable. Some fracture patterns merit special mention here. Bilateral sagittal plane fractures equate to a dissociation of the spinal column from the pelvis, whereas unilateral sagittal plane injuries generally preserve the weight-bearing axis on the uninjured side.[27,63–65] Sagittal injuries might combine with a transverse component as well, decreasing stability even more. Transverse injuries alone, when below the level of the sacroiliac joints, generally are of minimal consequence.

The fracture pattern and displacement often indicate whether neurologic deficit can be expected. Common indicators for nerve injury include displacement at the neural foramina and kyphotic segments, which can become focal areas of impingement. Intuitively, realigning and stabilizing the fractures with decompression of any neural elements should yield results superior to those achieved with nonoperative treatment; however, a review of the literature suggests that both patients treated operatively and those treated nonoperatively gain some neurologic recovery.[8,12,17,20,34,29,35,66] In our opinion, surgical decompression is warranted in the case of neurologic injury that is thought to be reversible.

NONOPERATIVE TREATMENT

Traditionally, treatment of sacral fractures included bed rest and then protected weight bearing. Bracing is sometimes used to further limit motion during the healing process. To treat fractures with displacement, traction can be used to regain some of the normal alignment. The amount of time spent recumbent depends primarily on the fracture type and stability; for stress fractures or insufficiency fractures, often, only a few days are required, with gradual return to activity. Unstable injuries, however, can equate to months of non–weight bearing. Because the down time required for stress fractures and insufficiency fractures is short, nonsurgical treatment is a viable modality for these fracture types. Often, even bilateral stress or insufficiency fractures can be mobilized early as long as the underlying metabolic conditions are managed appropriately.[39] Unilateral injuries that are minimally displaced can sometimes be mobilized early with protected weight bearing on the affected side.

When treating less stable injuries resulting from high-energy trauma, nonoperative treatment becomes less attractive. The disadvantages in these cases are as follows: (1) prolonged immobility, which places the patient at high risk of thromboembolism and poor pulmonary toilet, decubitus ulcers, and general deconditioning; (2) lack of direct decompression of the neural elements, which might be at risk for further damage; and (3) possibility of late deformity or instability with the development of progressive neurologic deficits.[67]

OPERATIVE TREATMENT

Operative fixation of sacral fractures is favored for widely displaced and unstable injuries, open injuries both externally and into the gastrointestinal tract, and injuries with neurologic deficits that seem reversible with decompression. Other reasons for operative treatment might include deformity that places the overlying skin at risk, either by direct pressure from bony fragments underneath the skin or prominences that can become areas likely to develop decubitus ulcers. The timing of fixation can be affected by the overall physiologic condition of the patient. In general, we think that early intervention provides the patient with stability for mobilization and allows timely decompression of the neural elements that might be compromised.

STABILIZATION TECHNIQUES

Surgical stabilization falls into three basic categories: open reduction and internal fixation of the sacrum, indirect sacral stabilization via pelvic ring fixation, and lumbopelvic fixation. Direct reduction and fixation of sacral fractures have been described, but their usefulness is limited because of the nature of the sacral bone. In general, the bone stock is suboptimal because of either fracture or osteopenia making screw purchase poor. In addition, this type of fixation generally is unable to withstand the forces generated across the lumbopelvic segment. One technique described by Roy-Camille et al.[27] that uses vertical plates placed lateral to the sacral foramina recommends 2 to 4 months of restricted weight bearing, which is no better than nonoperative treatment regarding early mobilization potential. This technique

generally is used only as an adjunct with other forms of fixation.

Pelvic ring stabilization can indirectly, and sometimes directly, stabilize the sacrum. Routt et al.[68] and Simonian and Routt[69] have shown that anterior pelvic ring stabilization alone is inadequate for unstable sacral fractures. Posterior stabilization is therefore required. Types of posterior fixation include tension band plating, iliosacral screws, and sacral bars. Although all three types provide fairly equivalent biomechanical stability to the posterior pelvic ring,[69] plating and sacral bars can be prominent and can lead to skin problems because of their superficial location and lack of overlying soft tissue coverage. Iliosacral screws are much less problematic in this regard, with plentiful soft tissue coverage. They generally are placed percutaneously after fluoroscopic reduction has been obtained. Iliosacral screws have been shown to be effective in stabilizing longitudinal fractures.[14,70,71] If used judiciously, iliosacral screws can be used to treat fractures that have a transverse component, such as the U or H fracture. In one series of patients, Nork et al.[72] showed that iliosacral screw fixation combined with a thoracolumbosacral-hip orthosis was an effective treatment modality for sacral fractures of this type. Some potential problems exist with iliosacral screw fixation for sacral fractures. Nerve injury from malpositioned screws and over-compression at the fracture site in comminuted sagittally oriented fractures causing nerve compression have been noted.[68] In addition, sacral angular deformity cannot be corrected and the neural elements cannot be decompressed. Although each of these types of posterior pelvic ring fixation provides stability to the sacral fracture and can allow early mobilization and decreased pain, none provides enough stability to allow full weight bearing.

Lumbopelvic fixation is by far the most stable of the three fixation types mentioned.[73] A dorsal approach like that used for posterior spinous procedures is used. The lumbar spine is instrumented with pedicle screw fixation in segmental fashion. The pelvis also is instrumented with screws placed from the posterior superior iliac spine to the anterior inferior iliac spine. The lumbar screws are then joined to the iliac screws by rods spanning the fractured sacrum and providing enough stability for full weight bearing.[74–77] All screws are placed with the use of fluoroscopic guidance, and the surgeon should have excellent understanding of pelvic anatomy. Correct placement must be assured, because misguided screws can have catastrophic results if they perforate the sciatic notch, the hip joint, or even the pelvic organs. The construct is low profile, avoiding some of the issues inherent with more subcutaneous constructs. But even with more soft tissue coverage, wound complications are seen in one of five patients. Because of the strength and stability of the lumbopelvic construct, patients generally can be mobilized without the need for bracing or weight bearing restrictions. The rods fatigue in many patients; however, by that time, stability generally has

been attained and most of these hardware failures are asymptomatic, making removal unnecessary.

No single type of fixation is suitable for all fracture types, and each is not exclusive of the others. In other words, a mix and match approach is sometimes the best when tailored to the specific fracture pattern. The previously mentioned U or H fractures are good examples of this. The sagittal fractures can be stabilized with iliosacral screws; however, that leaves the transverse component unstable. This can then be stabilized with plating, ending in a stable construct.[73,74] Perhaps the most important concept is that one must avoid a dogmatic approach, treating all fractures the same, but instead recognize what is unique about each fracture and tailor treatment accordingly using correct fracture fixation principles.

NEUROLOGIC DECOMPRESSION

Decompression of the neurologic elements is arguably one indication for operative management of sacral fractures. Sometimes decompression can be achieved indirectly, as in the case of impingement caused by kyphosis, which is corrected when the kyphosis is reduced. Indirect decompression, however, has limitations. Because the neurologic elements are not visualized directly, it is difficult to know whether the decompression was adequate, as function might have a delayed return. Also, reduction can be hampered by interposing tissues or, in the case of delayed treatment, organized hematoma can prevent reduction of the fractures or can actually increase the amount of nerve compression if the fractures are reduced.[78]

If indirect methods are not thought to be able to adequately decompress the compromised structures, direct methods should be used. Direct decompression can take many forms, but the basic idea includes visualization of the compressed neurologic structure and removal of any compressive lesion by reduction of misaligned fractures, excision, or both. Often, something as simple as a laminectomy can be very effective in removing compression from a compromised nerve root.[29] This can be performed at a single level or can be extended to include the entire sacrum. Laminectomy also can be combined with foraminotomy to gain more wide decompression. In general, a midline approach is recommended because the paramedian approach does not allow as wide an exposure of the dorsal sacrum.

At times, a compressive lesion is not correctible by simple decompression alone. Such is the case with widely displaced fractures or severely angulated fractures. In general, these situations require realignment of the sacrum with reduction of any large protruding fragments. Various reduction techniques have been described. We have found the following to be particularly useful. For angular sacral deformity, a Shanz pin can be used as a joystick to help reduce the fragments to each other. In the case of large displaced bone fragments, a

bone tamp might be useful in impacting the fragments back into place. Sometimes a combination of techniques provides the desired results.

With any decompression technique, epidural bleeding can become problematic; the surgeon should be adept at various hemostasis techniques. Traumatic dural tears also are common, requiring familiarity with dural repair and/or reconstruction techniques. The dural sac thins out in the sacrum, making repair challenging, and augmentation with patches, pericardial grafts, and/or DuraGen (Integra Life-Sciences, Plainsboro, NJ) often is required. Often, repair is not feasible because the tear involves the nerve roots distal to the end of the dural sac. In all cases of decompression, the surgeon should evaluate the overall stability of the sacrum and determine whether instrumentation is required to maintain stability and thus preserve the decompression and reduction.

CONCLUSION

Diagnosis and treatment of sacral fractures have changed dramatically with the advent of newer imaging techniques that allow more accurate diagnosis and understanding of fracture patterns. Diagnosis of neurologic injury has been improved in both the alert and the obtunded patient with neurodiagnostic studies. Treatment of sacral fractures continues to evolve with improving constructs and implants. The treating surgeon now has at his or her disposal options ranging from simple decompression to lumbopelvic fixation. The overall clinical picture of the patient is of paramount importance and should influence the treatment accordingly. As diagnostic and treatment modalities continue to evolve, it will be up to the surgeon to remain abreast of those changes and to adapt accordingly.

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Complications Related to the Surgical Management of Thoracolumbar Injuries

INTRODUCTION

Despite not being the topic of many papers, complications of the surgical treatment of thoracolumbar fractures are well known to the surgeons who strive to treat these conditions. This chapter aims to detail these complications, their treatment and, hopefully, by discussing them, facilitate their avoidance. Some complications are more subtle than the ones to be discussed. These have to do with the decision process made preoperatively to guide treatment; namely overtreatment and undertreatment.

It is not the intention of this chapter to re-discuss the surgical guidelines in approaching thoracolumbar fractures. Yet to discuss surgical complications without mentioned errors in the decision process is to miss the defining point in that complication. Surgical indications as they pertain to thoracolumbar fractures are a moving target—the goal is to strike the balance of overtreatment and undertreatment, to minimize the impact of the injury for the patient while restoring function. Stated another way, what is the least invasive procedure that will restore function to the patient in a safe and timely manner?

Undertreatment of thoracolumbar fractures is another way of saying underappreciated thoracolumbar fractures. These injuries often are referred to tertiary centers after late discovery at an outside institution, or are missed injuries on initial trauma surveys, and often provoke a knowing look from the receiving subspecialty physician. The sequelae from these delayed diagnoses, however, are generally negligible. Contrast to missed injuries of the cervical spine, neurologic

injury resulting from a missed thoracolumbar injury is extremely rare. Occasionally, an underappreciation of the magnitude of a thoracolumbar injury will manifest as a progressive deformity. Yet even this situation can generally be salvaged with a procedure that would not differ greatly from what would have been the appropriate intervention had the injury been diagnosed earlier.[1,2]

Overtreatment of thoracolumbar injuries is a more subtle phenomenon. The human body is remarkably resilient and has the ability to tolerate aggressive interventions and still provide reasonable outcomes. The more obvious of these would be surgical care for a minimally displaced thoracolumbar compression fracture. Surgical care may lead to an acceptable clinical outcome, even a satisfied patient, yet bracing would be the appropriate treatment.[3–8] More difficult to discern are differences in surgical choices. For example, an anterior L1 corpectomy with fusion from T12-L2 versus an anterior/posterior procedure from T12-L2 or a posterior fusion from T10-L3 might all be acceptable recommendations for a given L1 burst fracture but the surgical insult to the patient and potential long-term results might prove quite different.[9]

Surgical complications are part of surgical procedures yet, avoidance of many of them can be enabled by judicious decision-making prior to initiating care. Once the fracture has been deemed surgical, the approach will be anterior, posterior, or combined. The ensuing discussion is based on approach-specific complications.

SURGICAL COMPLICATIONS OF THE ANTERIOR APPROACH TO THORACOLUMBAR FRACTURES

INCISION/APPROACH RELATED
INCISIONAL PROBLEMS

The incision made in approaching the thoracolumbar spine often is the source of the most lasting complaints for the patient. A numbness about the incision, in particular distal to the incision often persists and is a function of transecting cutaneous nerves because of the oblique nature

of the incision. Improvement can be expected over time, yet minimizing the length of the incision can reduce the problem.

A pseudohernia can also result from this approach. The fibers of the external oblique, internal oblique, and transversalis have different orientations leading to transection of the fibers and denervation of the muscle. The resultant pseudohernia can be quite large and of true cosmetic concern. The smaller of these problems can be ignored with reassurance but a reconstruction with Marlex mesh is occasionally necessary. Attempting to stay higher and more lateral in the approach through the rib bed can minimize the dissection through the flank muscles.

DIAPHRAGM RUPTURE

Anterior approach to L1 or T12 fractures will generally necessitate detachment of the diaphragm to varying degrees. Minimizing detachment will minimize diaphragmatic complications but do not compromise visualization as diaphragmatic complications are rare. Some authors recommend leaving a lateral cuff of tissue laterally when detaching the diaphragm to later repair, whereas others, pointing out that innervation of the diaphragm is medially based, choose not to denervate a cuff of diaphragm and detach it directly from the chest wall. Critical for either approach is a secure closure.

SYMPATHETIC INJURY

The sympathetic chain to the lower extremities cascades along the anterolateral aspect of the spinal column. Mobilizing the chain is often necessary for exposure, thus potential for injury exists. The resultant erythema and warmth of the ipsilateral leg are often ignored by the patient and replaced by a complaint of coldness in the contralateral leg. Collateral innervation of the sympathetics generally leads to resolution of this complaint.

LYMPHATIC INJURY

In contrast to the segmental vessels, the lymphatics are quite difficult to dissect and control. Blunt dissection at the level of the disk with mobilization of the soft tissues from lateral to medial will minimize lymphatic disruption. Bipolar electrocautery is useful in controlling chyle leakage. If persistent leakage is suspected postoperatively, the patient should be made nil per os (NPO) to minimize chyle production. If the leakage is above the diaphragm, prolonged chest tube drainage may be needed. Rarely is re-exploration necessary.[10]

VASCULAR INJURY

It is generally recommended to approach the thoracolumbar spine from a left-sided approach. The surgeon thus avoids negotiating the bulk of the liver and also operates adjacent to the thick-walled aorta rather than the thinned-walled inferior vena cava. After radiographically confirming that the appropriate spinal level has been approached, the segmental vessels

should be identified and controlled. Once the segmental vessels have been controlled the great vessels may be mobilized to the contralateral side of the spine. Injury to the vessels occurs during dissection, diskectomy, or hardware placement and typically occurs in a sudden uncontrolled maneuver. Therefore, vigilance in protecting the vessels is critical. For example, a malleable retractor may be placed on the contralateral side of the spine at the level of the disk, thus protecting the vessels during diskectomy. As a general approach, always be aware of where the vessels are and work away from them.

Should an injury to the vessels occur, the surgeon must first tamponade, then obtain proximal and distal control. One should assess the injury, then proceed with primary repair. If a defect is created such that primary repair leads to vessel stenosis, a patched repair is necessary. The surgeon should never be hesitant to call for assistance from a vascular surgeon.

URETERAL INJURY

Additionally, on completion of hardware placement, the surgeon should remove all retractors and allow the local vasculature to return to its relaxed position. One should confirm that the newly placed hardware is not irritating the vessels as late vasculature injuries have been reported.[11–14] The ureter adheres to the posterior peritoneum and is most efficiently managed by mobilizing it with the posterior peritoneum rather than dissecting it out. Primary repair with stenting versus stenting alone should be performed with the guidance of a urologist. An unrecognized tear can typically be addressed by retrograde stenting, whereas an unrecognized transaction will require re-exploration.[14]

FRACTURE-RELATED
INADEQUATE DECOMPRESSION

One of the advantages of an anterior thoracolumbar approach to fracture or tumor is the ability to directly decompress the spinal column. However, the anterior approach does not guarantee that decompression will be satisfactorily achieved. Visualizing the superior and inferior endplates of the adjacent vertebral bodies and the base of the contralateral pedicle ensure an adequate decompression. Should a patient's symptoms not improve as a result of the surgery, the physician is obligated at some point to reassess the adequacy of the decompression. If persistent compression exists, the physician must then balance the risk of re-exploration and revision decompression with the potential benefit to the patient in terms of potential symptom relief.

INADEQUATE REDUCTION

Persistent deformity, typically kyphosis, after an anterior thoracolumbar approach can lead to persistent mechanical symptoms despite an adequate decompression. Thus obtaining anatomic alignment is a worthy goal. Maintaining and

re-establishing landmarks assist in regaining normal sagittal alignment. The anterior longitudinal ligament is rarely violated with fracture or tumor. By re-establishing tension in the anterior longitudinal ligament (ALL), anatomic height of the anterior column is obtained. Care must be taken to not over-distract posteriorly or the kyphotic deformity will persist. Adequate anteroposterior (AP) and lateral radiographs must be obtained in the operating room prior to closure to confirm sagittal and coronal alignment.

The relative contribution of a persistent kyphotic deformity to symptoms in the postoperative period is a much more difficult issue. Thirty degrees of persistent kyphosis in thoracolumbar fractures treated nonoperatively has been correlated to an increased incidence of back pain but extrapolating this number to the operatively treated patient may not be justified. Obtaining anatomic reduction intraoperatively removes deformity as a variable in postoperative pain.[15–19]

SURGICAL COMPLICATIONS OF THE POSTERIOR APPROACH TO THORACOLUMBAR FRACTURES

INADEQUATE DECOMRESSION

A disadvantage of the posterior approach to thoracolumbar fractures is the lack of direct visualization in performing and confirming an adequate decompression. The mechanism of reduction is characterized as relying on ligamentotaxis. Laminectomy with retraction of the neural elements to allow partial visualization has also been advocated. The surgeon must consider inadequate decompression when a patient does not make neurologic recovery. An axial study should be obtained and a decision made as to how to address the situation.[16]

PSEUDOARTHROSIS/HARDWARE FAILURE

The posterior approach to fixation of thoracolumbar fractures allows an excellent ability to obtain anatomic alignment of the spine. Operative positioning and transpedicular fixation maximize reduction capabilities. The posterior approach, however, does have a higher rate of nonunion, especially when a concomitant laminectomy or short construct is used. When alignment is restored but anterior column support is not re-established, a longer posterior construct is indicated.

Early hardware failure does not mandate surgical re-exploration. However, early hardware failure with increasing pain and deformity would require surgical treatment. Late hardware failure indicates a mature pseudarthrosis, but again, does not mandate surgical care. Pain, progressive deformity, and evolving neurologic loss are all surgical indications.

Salvage surgical care for failed thoracolumbar fractures often requires large surgical reconstructions. If posterior hardware is present in the face of marked deformity, a posterior exploration with hardware removal and osteotomy, followed by an anterior corpectomy with strut grafting and simultaneous posterior reinstrumentation and fusion may be necessary to adequately address the pathology.[20–33]

GENERAL COMPLICATIONS OF SURGICAL CARE OF THORACOLUMBAR FRACTURES

DURAL TEAR

Dural tears in the setting of thoracolumbar fractures are either injury induced or iatrogenic. A laminar split seen on a computed tomography (CT) scan with neurologic deficit has been cited as a surgical indication indicative of nerve root entrapment warranting exploration.

Injury-induced dural tears are rarely problematic. They are associated with a higher incidence of neurologic injury yet a, persistent dural leak is a rare occurrence. If the edges of the tear can be safely delineated, direct repair is warranted. This can be particularly difficult in anterior tears. If direct repair is not technically feasible then the surgeon must assess the options. If a massive tear is present the dural leak is low flow and typically self-limiting. Other options such as patching, fibrin glue, commercially available collagen matrix adjuncts, or a diverting lumbar drain may be considered.[34,35]

MISPLACED HARDWARE

Misplaced hardware in the thoracolumbar spine can have catastrophic results. In the anterior approach the challenge is in maintaining orientation of the vertebral bodies when placing vertebral screws. The surgeon must be mindful of both the location of the spinal canal so as to not direct the screws too far posterior, and the location of the great vessels to prevent placing the screws too long or anterior. Additionally, the corpectomy graft or hardware must be well seated in the superior and inferior endplates with an anatomic reduction to prevent retropulsion of the implant. Finally, intraoperative x-rays must be scrutinized for suspicious hardware placement and the potential problem addressed before leaving the operating room.

Hardware misplacement in the posterior approach to thoracolumbar fractures is also typically found in the misplacement of pedicle screws. Directing a screw too medial, too lateral, or too long all have negative implications. The surgeon may use surface landmarks, laminotomies, a pedicle probe, fluoroscopy, and other visual clues in the placement of pedicle screws. Final x-rays and a high index of suspicion are required.

If the patient develops a new deficit in the postoperative period, the surgeon must explain this finding. An axial study, such as a CT myelogram, can provide the occasionally

humbling answer. Re-exploration is indicated and communication critical.[4,13,15,20,23,31]

INFECTION

Wound infections after surgical care for thoracolumbar fractures are fortuitously rare, yet when they occur, aggressive care is necessary. Acute infections should be managed by debriding intraoperative cultures, retaining the hardware, ensuring adequate soft-tissue coverage, and providing definitive antibiotic coverage. Antibiotics should be continued until at least normalization of C-reactive protein, white blood count (WBC), and sedimentation rate. If infection recurs the treatment plan should be repeated. Consideration should be given for suppressive antibiotics in this setting, with late hardware removal and repeat debridement once the fusion has matured.[36]

Late wound infections are more easily treated but often more difficult to recognize as they can be quite indolent. If a patient reports late onset pain the differential should include both pseudarthrosis and occult wound infection. The ensuing workup of laboratory tests and CT scans may be inconclusive and the surgeon is left with the debate as to whether or not to re-explore. If infection is found, culture, hardware removal, and aggressive debridement with postoperative antibiotics will generally yield an excellent result.

THROMBOSIS

Many medical complications can occur during the perioperative treatment of thoracolumbar fractures and the surgeon must be vigilant in his or her ongoing postoperative assessment. Deep venous thrombosis and pulmonary embolism bear further discussion both for the life-threatening nature of this complication and the increased incidence in the trauma population. Mechanical prophylaxis is indicated. Prophylactic placement of intravenous (IV) filters in patients who, because of comorbidities, will be treated with prolonged bedrest should be considered versus medical prophylaxis. Doppler studies can confirm suspected deep venous thrombosis and a CT scan can confirm pulmonary embolism. Medical treatment with anticoagulation therapy is indicated for confirmed thrombosis.[37]

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Mechanisms of Injury in the Sacral Spine: Concepts, Pathomechanics, Classifications, Instability, and Clinical Applications

GENERAL CONCEPTS

DEMOGRAPHICS

Sacral fractures occur less often in children than in adults and usually are less displaced and more amenable to nonoperative treatment in the pediatric population. They occur primarily in association with pelvic fractures. Pelvic fractures occur in approximately 3% of children sustaining high-energy injuries, whereas sacral fractures occur in approximately 0.16% of all pediatric trauma patients.[1,6] Mortality rates range from 1.4% to 14% of patients with pelvic fractures,[1–5] mainly related to associated injuries.[1,3,4,6] Although some studies suggest that pediatric sacral fractures tend to affect older adolescents,[7] others have shown that they can occur evenly throughout a broad age range.[6] Although sacral fractures occur in only approximately 5% of children with pelvic fractures (as opposed to approximately half of adults with pelvic fractures), they are present in approximately 60% of children with pelvic fractures who have neurologic deficits.[6,8]

Much like in adults, sacral fractures in children involve primarily the sacral ala[6] or the neuroforaminal region because these areas tend to be the weakest in the sacrum. The distribution of sacral fractures in children is skewed heavily toward Denis zone 1 injuries, which have been reported to occur in 75% of patients, with the remaining 25% of injuries being evenly distributed between zone 2 and 3 injuries.[6] Sacral fractures that result in spinopelvic dissociation (i.e., zone 3 sacral U-type fractures and their variants) are rare injuries in both the pediatric and adult populations. However, whereas busy trauma centers have reported case series describing a considerable number of adult patients with these injuries,[9,10] their presence in the pediatric population has been described in few individual case reports.[11,12]

When sacral fractures do occur in children, they generally are nondisplaced or minimally displaced.[6,7] Greenstick fractures of the sacrum also have been reported as occurring in children.[13] Most sacral fractures in children can therefore be treated nonoperatively. Nevertheless, unstable sacral fractures do occur in children, and the principles behind their treatment are similar to those for adults (Fig. 40-1).[14] Treatment of sacral fractures largely is contingent on pelvic ring stability; more than 90% of pelvic fractures in children,[3] particularly those with open triradiate growth plates and the opportunity for extensive remodeling, do not require surgical intervention.

As with the adult population, delayed and missed diagnoses are relatively common problems associated with pediatric sacral fractures. Computed tomography (CT) of the pelvis is recommended for children who have sustained high-energy injuries and who have ecchymoses of the lower back or buttocks, sacral point tenderness, pelvic instability, radiculopathy, and/or bowel/bladder abnormalities.[6] Also analogous to the situation with adults, the increasingly routine use of pelvic CT for evaluating trauma has likely diminished the number of missed sacral fractures.

Sacral fractures more commonly occur in the adult population. They occur in approximately 5% of all adult trauma patients and in approximately half of adults with pelvic fractures.[8,15] Adults are twice as likely as children to sustain pelvic fractures in a motor vehicle collision and 15 times as likely to fracture the pelvis from a fall of 15 feet or greater.[8] Moreover, whereas sacral fractures might occur in adults as a result of low-energy trauma because of underlying metabolic bone disease, such as osteoporosis, sacral fractures in children almost exclusively are the result of high-energy injuries, such as motor vehicle collisions, automobile versus pedestrian trauma, and falls from a height of 25 feet or more.[6,11,12]
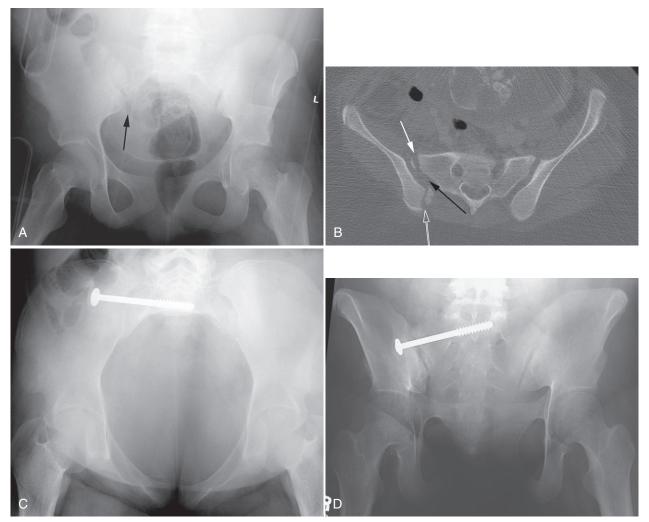
Fig. 40-1 Iliosacral screw fixation for a child with a zone II sacral fracture with pelvic instability. AP view of the pelvis *(A)* and axial CT image *(B)* show pelvic asymmetry with a displaced zone II sacral fracture. Postoperative inlet *(C)* and outlet (*D*) views of the pelvis show fixation of a symmetrically aligned pelvis with the use of iliosacral screws. The fundamentals of treating sacral fractures associated with pelvic instability do not differ greatly between adults and children.

ANATOMIC CONSIDERATIONS

In addition to serving as the posterior bridge between the iliac wings, the sacrum is the structure that joins the spine to the lower extremities through the sacroiliac joints. The triangular cross-section of the sacrum at the level of the sacroiliac joints allows it to function as the "keystone" that supports the posterior pelvic arch, in conjunction with its dense ligamentous attachments to the iliac wings. The sacral alae constitute the lateral parts of this base, which articulate with the iliac wings through the sacroiliac joints. The upper two sacral vertebrae form the auricular facet of the sacroiliac joint. The lower sacral vertebrae and the coccyx are nonarticular but contribute to pelvic stability by supplying the attachments for the sacrotuberous and sacrospinous ligaments. In addition to its key role in posterior pelvic ring stability, the sacrum and its surrounding ligaments also form the integral foundation for the structural

integrity of the lumbar spine and the protective conduit for major neurovascular structures supplying the pelvis and lower extremities.

PATHOMECHANICS

In a healthy, active person, high-energy injury mechanisms are required to cause a fracture of the sacrum.[16] In keeping with the dual structural role of the sacrum, as noted previously, injury to the sacrum can impact stability of the posterior pelvic ring, the spinopelvic junction, or both. In addition, direct-injury mechanisms, such as forces applied to the buttocks, generally result in low, transverse sacral fractures that do not affect either pelvic or spinopelvic stability. Indirect mechanisms, with forces transmitted through the lumbosacral spine and lower extremities across the sacroiliac joints, however, result in shear fracture patterns with

vertical and possibly high transverse orientations that can compromise spinopelvic and pelvic ring stability.

TREATMENT

Most sacral fractures are relatively stable nondisplaced or minimally displaced injuries that require only a 1- to 3-month period of restricted weight bearing. This is particularly true of fractures in children. Operative intervention is recommended for injuries with unacceptable displacement and injuries that impact either pelvic ring or spinopelvic stability. Operative treatment paradigms for sacral fractures with mainly posterior pelvic instability (e.g., vertical sacral fractures) have been fairly well established and primarily involve the use of iliosacral screw fixation, with or without open reduction.[17] Regarding complex multiplanar sacral fractures with vertical and transverse components (e.g., spinopelvic dissociations), treatment algorithms are not as well established. The wide variability in specific injury types and the relatively low incidence of these injuries have impeded the development of a generalizable treatment paradigm. Newer spinopelvic stabilization techniques, however, carry considerable promise in their ability to reconstruct the anatomic relationships between the lumbosacral spine and the pelvis that have been disrupted by these injuries.[9,10]

PATHOMECHANICS

Sacral fractures can be vertically oriented, transversely oriented, or complex with vertical and transverse components (the so-called U- or H-type fractures). Vertical fractures mainly affect posterior pelvic stability, usually without influencing spinopelvic relationships. However, involvement of the L5-S1 facet joint, even in vertically oriented sacral fractures, can cause lumbosacral instability.[18] Regarding transverse fractures, an important distinction must be made between isolated transverse sacral fractures, which usually occur caudal to the sacroiliac joints and do not compromise the stability of either the pelvic ring or the spinopelvic junction, and transverse fractures that occur between the sacroiliac joints, have associated vertical sacral fractures, and might affect both pelvic ring and spinopelvic stability.

Vertically oriented sacral fractures can be caused by lateral compression that causes impaction of the cancellous bone of the sacrum, by tension failure from an "open book" pelvic injury, or by shear forces propagated across the sacroiliac joints from the lower extremities. Such shear forces are the result of oppositely directed forces applied to the lumbosacral spine and the lower extremities.[19] High-energy fractures, especially those caused by falls from a height, might involve bilateral vertical fractures joined by one or more horizontal fracture lines, resulting in the so-called sacral U- or H-type fracture patterns.[16] As with vertical sacral fractures alone, the biomechanics of these sacral U-fracture variants involve shear loading that results from transmission of the weight of the torso across the lumbosacral spine, counteracted by the lower extremity ground reaction forces propagated in the opposite direction across the acetabula

and sacroiliac joints.[19] As mentioned previously, the transverse fracture component of these complex sacral fractures usually involves the upper sacral segments lying between the sacroiliac joints, thus compromising spinopelvic stability. The injury pattern, also known as *spinopelvic dissociation,* results in two primary fracture fragments: one consisting of the spine and central sacral fragment and the other consisting of the pelvis and peripheral sacral fragment. The deforming shear forces across this injury promote "shortening" of the sacral fracture, which effectively results in anterocaudal displacement of the lumbar spine and its attached upper central sacral fragment into the pelvis (Fig. 40-2). In a true sacral U-fracture, the vertical fracture lines are limited to the upper central sacrum, whereas the peripheral sacrum remains intact, in which case posterior pelvic stability is not compromised. If the vertical fractures extend more caudally, as with sacral H-fractures, posterior pelvic instability can coexist with spinopelvic instability (see Fig. 40-2).

These shear mechanisms result more often in sacral fractures than in sacroiliac joint dislocations because the sacral
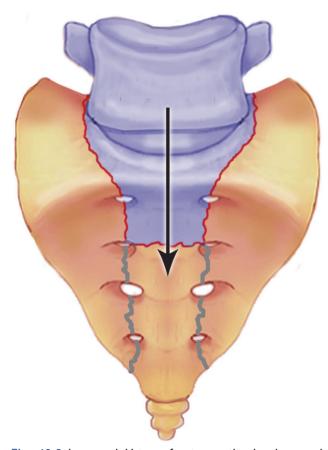

Fig. 40-2 In sacral U-type fractures, the lumbosacral spine is essentially detached from the peripheral part of the sacrum and the pelvis, resulting in spinopelvic instability. The lumbosacral spine bends toward kyphosis and anterocaudal translation into the pelvis. In situations in which the longitudinal or vertical fracture lines exit the sacrum caudal to the sacroiliac joints (gray fracture lines), the resulting sacral H-type fracture pattern would also result in posterior pelvic instability.

bone is weaker and more prone to failure under shearing loads than is the stout sacroiliac ligament complex. For this reason, vertical sacral fractures, regardless of whether they are unilateral, bilateral, or occurring in association with transverse fractures, preferentially involve the ala and the neuroforamina because they represent areas of structural weakness. Biomechanical evaluation of injuries to the pelvis also has shown that the location of the fracture seems to be related to the impact energy to which the pelvis is subjected.[20] Lower energy loads result primarily in fractures of the acetabulum and ilium, including crescent fractures. Higher energy impacts result in all three types of sacral fractures as classified by Denis et al.,[21] with a propensity for zone 1 and 2 injuries.

Isolated transverse fractures usually occur as the result of direct trauma, such as a fall onto the buttocks with impact on the caudal sacrum. This mechanism results in a kyphotic bending movment, which has a fulcrum just below the sacroiliac joints and which therefore usually causes a fracture at the S3 level. These fractures are not particularly common in adults and are even more unusual in children.[22]

In contrast to sacral fractures that occur as a result of high-energy mechanisms of injury, the cause of sacral insufficiency fractures often is not identifiable or is a trivial event, such as a fall onto the buttocks from a standing or sitting position. Insufficiency fractures occur in weakened bone and typically occur in postmenopausal women because of osteoporosis, although younger patients with metabolic bone disease[23] or even pregnant or postpartum women[24] also are susceptible. Other risk factors for osteoporosis might also be present, such as chronic corticosteroid use or a history of radiation therapy to the pelvis. The common link is the presence of pathologic bone, whatever the specific cause. The presenting symptoms often are vague and often consist of poorly localized low back pain that might be exacerbated by sitting and standing. These fractures typically are vertically oriented and located in the sacral ala, adjacent to the sacroiliac joint. The reason that sacral ala involvement is typical of insufficiency fractures is that the sacral alae seem preferentially affected by metabolic bone disease, which results in severe osteopenia within the ala. Insufficiency fractures also might have a transverse component, resulting in more complex U-and H-fracture variants (Fig. 40-3). Because of the
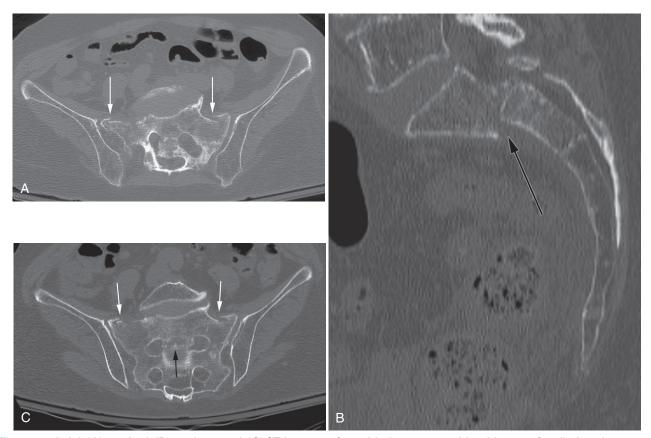

Fig. 40-3 Axial *(A),* sagittal *(B),* and coronal *(C)* CT images of an elderly woman with a history of radiation therapy to the pelvis that had been experiencing worsening low-back pain for several months and an inability to sit or stand. The CT images demonstrate the typical sacral U insufficiency fracture pattern longitudinally *(white arrows),* adjacent to the sacroiliac joints, along with a transverse sacral fracture *(black arrows).* Sacral-insufficiency fractures are a commonly undiagnosed reason for low back pain. After treatment with lumbopelvic fixation, the patient was immediately able to sit and bear weight with markedly reduced pain, and she subsequently healed her fracture uneventfully.

predisposition of these patients to osteoporotic fractures, a high likelihood of insufficiency fractures at other sites is present, most commonly the pubis and thoracolumbar vertebrae. Neurologic deficits are uncommon in most series; however, cauda equina dysfunction has been reported and neurologic status must be carefully considered.[25–27]

Mechanical factors related to the transfer of forces from the lumbosacral spine to the pelvis also can cause sacral insufficiency fractures in patients who are predisposed because of osteoporosis. Specifically, unilateral sacral insufficiency fractures have been identified on the concave side of lumbar scoliosis.[26] Pelvic stress concentration as the result of lumbosacral spine fusion also has been described, and sacral insufficiency fractures caudal to previous lumbar fusions also occur, although infrequently. These fractures probably represent failure of the sacrum to withstand forces concentrated there as a result of the large cephalad lever arm.[28–30]

Regarding the distribution of sacral insufficiency fractures among age groups, these fractures do not occur with any degree of frequency in the pediatric population unless in the setting of severe metabolic bone disease. Sacral insufficiency fractures are particularly common among the elderly population because of osteoporosis.

Stress fractures of the sacrum, as opposed to insufficiency fractures, occur in bone that is not weakened by a pathologic process but in situations in which a high activity level causes repetitive stress that exceeds the reparative ability of normal bone. Therefore, these fractures can occur in younger patients, particularly in high-demand persons, such as endurance athletes and military recruits. Although far less common in the pediatric population than in adults, several cases of stress fractures in children and adolescents have been reported.[31–34] Sacral stress fractures seem to preferentially involve the alae.[31–34] As with patients with insufficiency fractures, these patients might present with vague and nonspecific low back pain. Treatment usually involves moderation of activities.

In some instances, the cause of low-energy sacral fractures is related to both repetitive stress and insufficiency. As an example, sacral fractures in young female athletes with amenorrhea might result from both overuse and osteopenia.

CLASSIFICATION

In 1988, based on a series of 236 sacral fractures, Denis et al.[21] formulated a simplified anatomic classification that correlates fracture location with the incidence of neurologic injury. This classification divides the sacrum into three zones (Fig. 40-4). Zone 1 (alar zone) fractures remain lateral to the neuroforamina throughout their course. Zone 2 (foraminal zone) fractures are located at the transition area between the sacral ala and body and involve one or more neuroforamina while remaining lateral to the spinal canal. Zone 3 (central zone) fractures involve the spinal canal. The incidence of neurologic injury increases as fractures are more centrally located. Accordingly, zone 1 fractures have been reported to

Fig. 40-4 Classification of sacral fractures as described by Denis et al.[21] Zone 1, fracture lateral to neuroforamina; zone 2, fracture involves neuroforamina but not the central canal; and zone 3, fracture involves the central canal.

have a 5.9% incidence of neurologic injury, primarily to the L5 nerve root as it coursed over the ala. Zone 2 fractures have been reported to have a 28.4% incidence of associated neurologic injury, consisting of primarily unilateral lumbosacral root deficits occurring as a result of either foraminal displacement causing impingement on the exiting nerve root or of the "traumatic far-out syndrome" in which that L5 nerve root is caught between the L5 transverse process and the displaced sacral ala. Zone 3 fractures had a 56.7% incidence of neurologic injury resulting from injury at the level of the spinal canal. Because of the potential for bilateral sacral root injury in fractures involving the spinal canal, 76.1% of neurologic deficits seen with zone 3 fractures were noted to involve bowel, bladder, and sexual dysfunction.

Because of their high potential for associated neurologic injury and spinal column instability, Denis zone 3 fractures have been specifically studied by several investigators. Early case reports often characterized the injury pattern of upper sacral fractures with transverse components as consisting solely of a transverse fracture, possibly because of imaging limitations. CT imaging, however, demonstrates that "transverse" fractures of the upper sacrum have more complex fracture patterns. The majority of these injuries are now understood to consist of a transverse fracture of the sacrum with associated longitudinal or "vertical" injury components, usually in the form of bilateral transforaminal fractures that extend rostrally to the lumbosacral junction—the so-called U-fracture. Variations in fracture line propagation include the H-, Y-, and lambda fracture patterns, among others (Fig. 40-5). Whether
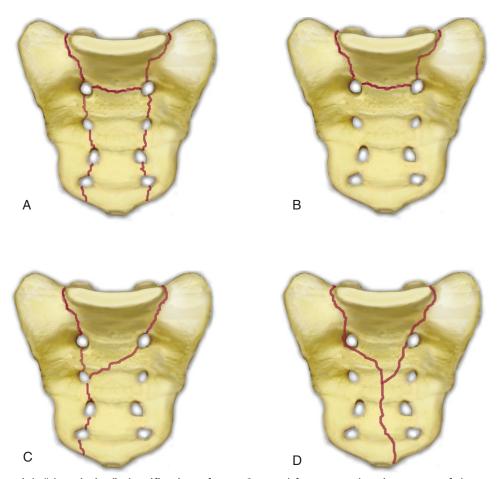
Fig. 40-5 Colloquial, "descriptive" classification of zone 3 sacral fractures, showing some of the possible complex sacral fracture patterns. *A,* H-fracture. *B,* U-fracture. *C,* Lambda fracture. *D,* Y-fracture.

or not the vertical fracture lines extend caudal to the sacroiliac joint and exit the caudal aspect of the sacrum are important in determining posterior pelvic ring stability.

The transverse fracture component of multiplanar zone 3 injuries has been further characterized based on its angulation and translation. In 1985 Roy-Camille et al.[16] described 13 patients with transverse sacral fractures, 11 of whom were the result of attempted suicide by jumping. They classified the fractures as type I, flexion deformity of the upper sacrum without translation; type 2, flexion deformity with posterior displacement of the upper sacrum on the lower sacrum; and type 3, anterior displacement of the upper sacrum with either no angulation or with an extension deformity (Fig. 40-6). Based on cadaveric studies, they hypothesized that types 1 and 2 were caused by the lumbar spine being in a flexed position at the time of impact, whereas type 3 fractures were caused by the lumbar spine and hips being in extension at the time of impact. Strange-Vognsen and Lebech,[35] subsequently reported the type 4 injury, a case of comminution of the upper sacrum without significant angulation or translation, that they attributed to an axial loading mechanism resulting from neutral lumbosacral spine position at the time of impact (see Fig. 40-6).

Even in the absence of a transverse fracture line, sacral fractures can result in spinal column instability. Isler[18] described variations of zone 2 longitudinal sacral fractures through the neuroforamina that may result in L5-S1 motion segment instability resulting from associated L5-S1 facet joint disruption. Injuries with the fracture line lateral to the S1 articular process are not associated with instability of the lumbosacral articulation because the L5-S1 articulation remains continuous with the stable fracture component of the sacrum (Fig. 40-7). A fracture that extends into or medial to the S1 articular process, however, may disrupt the associated facet joint and potentially destabilize the lumbosacral junction. Complete displacement of the facet joint can cause a locked facet joint making sacral fracture reduction difficult with closed methods alone (Fig. 40-8). Facet disruption may also cause post-traumatic arthrosis and late lumbosacral pain.

Gibbons et al.[36] studied the association between the Denis classification and patterns of neurologic injury and devised a classification system for the severity of neurologic impairment caused by fracture of the sacrum. Neurologic injuries were classified as 1—none, 2—lower extremity paraesthesias
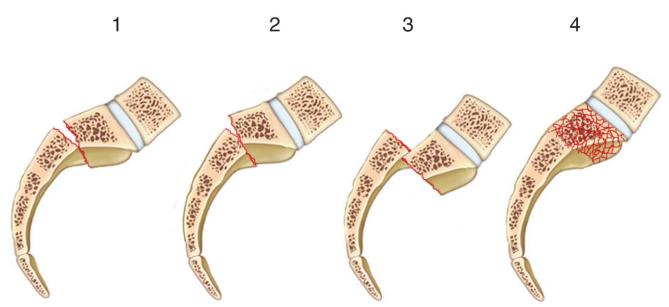
Fig. 40-6 Subclassification of Denis zone 3 fractures according to sagittal plane alignment by Roy-Camille et al[16]. and Strange-Vognsen and Lebech[35]. Type 1, flexion injury without translational displacement of the sacrum; type 2, flexion injury with translational displacement; type 3, extension injury of the sacrum; and type 4 (Strange-Vognsen), axial-loading injury with segmentally comminuted S1 body.
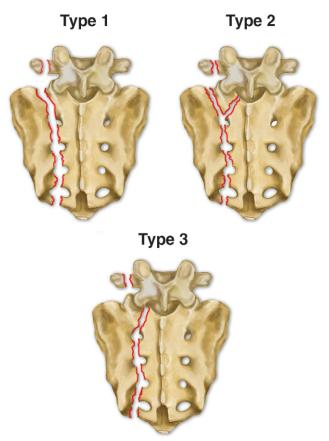

Fig. 40-7 Isler classification of lumbosacral junction instability associated with Denis zone 2 sacral fractures, based on the location of the fracture relative to the L5-S1 facet joint[18].
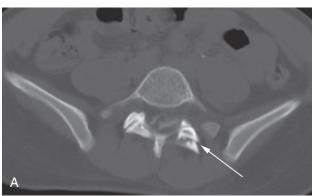
only, 3—lower extremity motor loss ± paraesthesias, bowel and bladder intact, and 4—bowel and/or bladder dysfunction (Table 40-1). They also noted that significant neurologic deficits are rare in transverse sacral fractures below the S4 level.
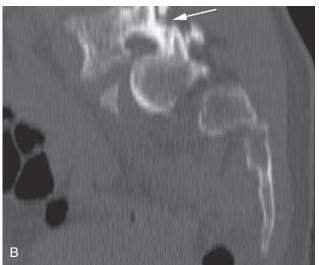
INSTABILITY AND CLINICAL APPLICATIONS

GENERAL TREATMENT CONSIDERATIONS

Vertically oriented sacral fractures that result in unacceptable pelvic ring instability or malalignment require early reduction and fixation regardless of whether they occur in children or adults. The correction of late deformities resulting from malunion or nonunion is more complex and carries a much higher morbidity than does early treatment. In children, the potential also exists for fracture-related growth abnormalities and leg-length discrepancy, which can further complicate the clinical picture. In light of this issue, early restoration of anatomic pelvic anatomy is likely to provide the most favorable possible long-term outcome.

Although uncommon, sacral U- or H-type sacral fracture-dislocations that result in spinopelvic dissociation with neurologic impairment occur almost exclusively in polytraumatized adults. Only a small number of case reports have described these injuries in children.[11,12] Treatment of these high-energy injuries has traditionally been nonoperative by default because of the absence of effective surgical treatment alternatives for the reduction and
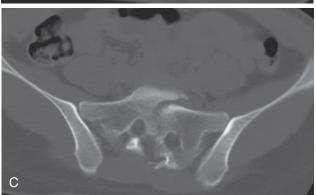
Fig. 40-8 *A* and *B,* Axial and sagittal CT images respectively, of the pelvis of a young woman who was involved in a motor vehicle collision and sustained a zone 2 sacral fracture. Note the dislocated L5-S1 facet dislocations *(white arrow). C,* Because the lateral sacrum is continuous with the displaced S1 facet joint and is therefore locked in a posteriorly displaced position, realignment of the sacral fracture cannot be accomplished without open reduction of the facet joint.

TABLE **40-1**   Gibbons Classification of Cauda Equina Impairment

TYPE	NEUROLOGIC DEFICIT
1	None
2	Paresthesias only
3	Lower extremity motor deficit
4	Bowel/bladder dysfunction

neutralization of these complex spinopelvic instability patterns. However, if left untreated, whether by intention or in the common scenario of a missed diagnosis,[16,21,37,38] painful deformity or progressive neurologic dysfunction may occur. As with the treatment of vertical sacral fractures, late corrective surgery is more complex and generally associated with worse outcomes. Therefore, early realignment and fixation of the unstable spinopelvic junction is thought to provide the best possible environment for safe, early mobilization of the polytraumatized patient while protecting against progressive soft tissue or neural injury and late fracture displacement.

Neurologic improvement after sacral fractures with spinopelvic instability patterns has been reported to approach 80%.[9,10,39,40] Several case series have described better neurologic outcome with surgical treatment, particularly in patients with bowel and bladder impairment.[21,36,41] However, these studies have significant shortcomings that limit the applicability of their conclusions. The influence of operative intervention on nerve root recovery has been difficult to discern. In a postmortem study of 42 individuals with posterior pelvic ring disruptions, Huittinen[42] studied the incidence and anatomical basis of lumbosacral nerve injuries, thus shedding additional light on these severe injuries and on potential treatment options. He noted that traction injuries comprised 53% and root disruption 38% of all nerve injuries. Compression injuries, the most amenable to surgical intervention, constituted only 20% of the nerve injuries. This study suggested that the ability of surgical intervention to improve neurologic function after sacral fractures may be limited. However, because of the considerable redundancy in sacral root innervation and the need for only unilateral sacral root function for certain vital functions, improvement of function in relatively few of the injured roots may translate to tremendous functional gains. Moreover in addition to addressing root compression, by achieving sacral fracture realignment and stabilization, surgery may also facilitate the recovery of roots injured by a traction mechanism. These details, among others, may explain

the apparent discrepancy between the low likelihood of neurologic recovery that would be anticipated from the previously mentioned anatomic findings and the fact that recovery of sacral root function tends to occur in more than 80% of patients.

Unlike cauda equina syndrome caused by acute lumbar disk herniation in which urgent decompression within 24 to 48 hours is generally advocated, the timing of sacral root decompression for fracture has not been determined. Because of the highly dissimilar injury mechanism and patient populations, the recommendations for surgical intervention in patients with cauda equina syndrome owing to lumbar disk herniation cannot be extrapolated to patients with sacral fractures. However, improved neurologic recovery may be a potential benefit of early decompression of compromised neural elements. These benefits must be weighed against increased risks of hemodynamic instability, severe blood loss, and wound infection in the physiologically compromised sacral fracture population. Indications for emergent surgical intervention include deteriorating neurologic status, dorsal soft-tissue compromise from displaced fractures, and the presence of an open fracture. It appears that most patients can be effectively treated surgically within a 48-hour to 2-week window. Prolonged delay in patients with canal encroachment and concordant neurologic deficit, however, may negatively influence neurologic recovery.[21,43,44]

BIOMECHANICS OF FIXATION OPTIONS
VERTICAL SACRAL FRACTURES (POSTERIOR PELVIC RING INSTABILITY)

In terms of treatment of posterior pelvic ring instability (vertical sacral fractures) available, posterior internal fixation options include a spectrum of percutaneous and open techniques such as iliosacral screws, iliac bars, tension band-plate fixation, and lumbopelvic fixation methods. The goals of fixation with this injury pattern are to neutralize the shear forces that promote shortening of the affected hemipelvis and the three-dimensional rotational forces that promote pelvic malalignment. The literature regarding comparative biomechanical testing of posterior pelvic ring fixation techniques is varied and somewhat inconclusive. Generally speaking, however, there appears to be little difference in effectiveness between the use of one versus two iliosacral screws, anterior sacroiliac plates, transiliac or sacral bars, and a tension band plate.[17,45–50]

The use of lumbopelvic fixation, however, has been shown to provide significantly improved fixation of vertical sacral fractures (Fig. 40-9). Schildhauer et al.[51] conducted a biomechanical comparison of triangular osteosynthesis (lumbopelvic fixation plus iliosacral screw) versus standard iliosacral screw osteosynthesis alone for unstable transforaminal (zone 2) sacral fractures. They found that triangular osteosynthesis provided far less fracture displacement for a given load, and
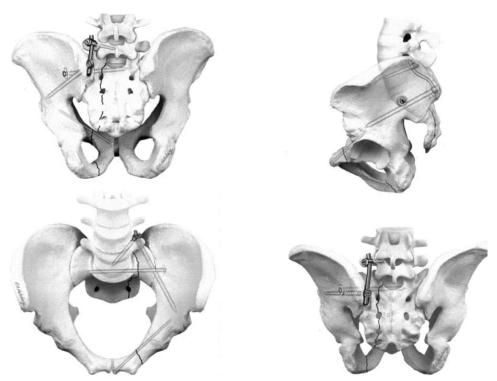

Fig. 40-9 Triangular osteosynthesis for the treatment of vertical sacral fractures. (From Schildhauer TA: J Ortho Trauma 17:22–31, 2003.)

protected against catastrophic failure of fixation as compared with iliosacral screw fixation alone. They concluded that triangular osteosynthesis for unstable transforaminal sacral fractures provides significantly greater stability than iliosacral screw fixation under in vitro cyclic loading conditions. Whether this enhanced stability routinely warrants the somewhat more invasive approach to stabilizing zone 2 sacral fractures remains a matter of controversy.

Variations of the triangular osteosynthesis concept have also been reported. Sar and Kilicoglu[50] reported on the use of screws placed in the S1 pedicle and the ilium (Fig. 40-10) for the treatment of zone 1 sacral fractures with functional sacroiliac joint instability. Vertical stability of the new technique was compared with anterior double-plating and iliosacral screw fixation techniques, with favorable results.

In summary the biomechanical literature comparing various posterior pelvic fixation methods in the treatment of vertical sacral fractures shows repeated use of several different fixation modalities, such as iliosacral screws, anterior sacroiliac joint plates, tension band plates, and iliac/sacral bars. Few differences in stability between these forms of fixation can be inferred, however, and each of these biomechanical investigations uses different loading conditions and methodology to assess fixation stiffness, further compromising the clinical applicability of these studies. However, the study by Schildhauer et al.[51] suggests that lumbopelvic fixation methods offer significantly greater stability than iliosacral screw constructs alone in the treatment of zone 2 sacral fractures and may therefore warrant more routine use.

SPINOPELVIC DISSOCIATION

Highly displaced multiplanar sacral fracture-dislocations that result in spinopelvic dissociation and are usually accompanied by cauda equina deficits are poorly understood and continue to pose diagnostic and treatment challenges. Because they are generally comprised of a transverse sacral fracture component with associated bilateral longitudinal fracture components, these fractures result in dissociation of the spine and upper central sacral segment from the pelvis and peripheral sacral fragments, giving rise to the term "spinopelvic dissociation" (Fig. 40-11).[51,52] The emergence of segmental spinal fixation techniques that can incorporate the iliac wings has allowed the opportunity to achieve reduction and stabilization of fracture-dislocations involving the lumbosacral region, along with aggressive neural decompression, while permitting early mobilization without supplemental bracing.[53] Although iliosacral screws offer an elegant percutaneous method of posterior pelvic ring fixation, they require a virtually anatomic reduction and the absence of anatomic variants. In addition, they have limited application in patients with either poor bone quality or extensive comminution of the sacrum, which are commonplace with spinopelvic instability patterns. Furthermore, although the perpendicular orientation of iliosacral screws is favorable for fixation of longitudinal sacral fractures, percutaneous iliosacral screw fixation provides little or no load-bearing capacity to a compromised spinopelvic junction.[50]
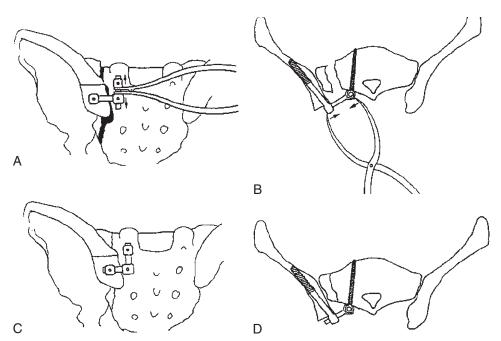

Fig. 40-10 Pediculoiliac fixation for a Denis zone 1 sacral fracture. *A,* Correction of vertical displacement using a distractor. *B,* Correction of lateral displacement using a compressor. Final posterior *(C)* and axial *(D)* views of the pelvis. (From Sar: J Orthop Trauma 17:263, 2003.)
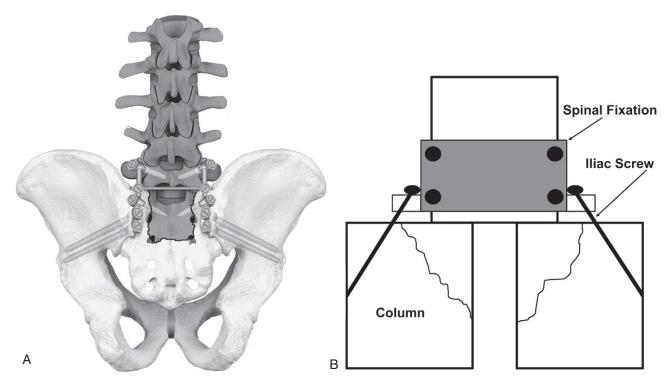
Fig. 40-11 Lumbopelvic fixation for stabilization of sacral fractures that result in spinopelvic dissociation. *A,* A schematic representation of the lumbosacral spine and pelvis shows how with sacral U fractures, lumbopelvic fixation secures the "rostral" fracture fragment *(shaded),* which consists of the entire spine and central upper sacrum, to the "caudal" pelvic fracture fragment, which consists of the peripheral sacrum and pelvis. The basic idea consists of bypassing the injured sacrum by achieving fixation in the lumbosacral spine and iliac wings. *B,* This construct provides a broad, solid foundation for fixation of the spine to the pelvis and unloads the S1 segment's keystone-type weight-bearing function at the lumbopelvic junction until healing has occurred.

Lumbopelvic fixation is designed to counteract the commonly misunderstood pattern of spinopelvic instability that occurs with sacral U-type fracture variants. Securing the lumbosacral spine to the iliac wings with segmental spinal fixation neutralizes the deforming forces that tend to otherwise result in anterocaudal displacement of the spine relative to the iliac wings. This is accomplished by gaining solid fixation in both major components of the fracture—the lumbosacral component and the pelvic component. In so doing, the relationship of the lumbosacral spine relative to the pelvis can be restored and maintained. In fractures in which the vertical components extend throughout the entire course of the sacrum, the resulting posterior pelvic instability is also neutralized because the iliac wings are secured to each other via the lumbosacral spine (see Fig. 40-11). Cross connectors can further stabilize the hemipelves to each other. This method of fixation is particularly valuable because sacral anatomic constraints, poor bone quality, and extensive comminution have made direct sacral fracture fixation difficult to achieve with more traditional plate and screw constructs.[51]

Biomechanical analysis has confirmed that segmental lumbopelvic instrumentation, a method derived from the Galveston technique of anchoring spine constructs caudally to the ilium, is among the most stable methods of posterior spinopelvic fixation.[51,54,55] This technique is particularly well suited to the treatment of complex sacral fractures with spinopelvic instability, because it unloads the area of injury by mimicking the normal load transfer from the acetabula across the sacroiliac joints to the lumbar spine (see Fig. 40-11).

A CT-based evaluation of pelvic anatomy by Schildhauer et al.[55] has helped determine the preferred trajectories for iliac screw placement as well as the screw length and diameter that are typically accommodated by the average male and female pelvis. Unfortunately, no such data are available for pediatric patients, although appropriate screw sizes can be determined in children by evaluation of the CT scan on a case-by-case basis in the few situations in which lumbopelvic fixation is required (Figs. 40-12, 40-13).[14]

SUMMARY

Overall, the principles behind the injury pathomechanics, classification, and treatment of sacral fractures are similar in children and adults. However, the likelihood of fracture to the sacrum, particularly displaced or unstable fractures
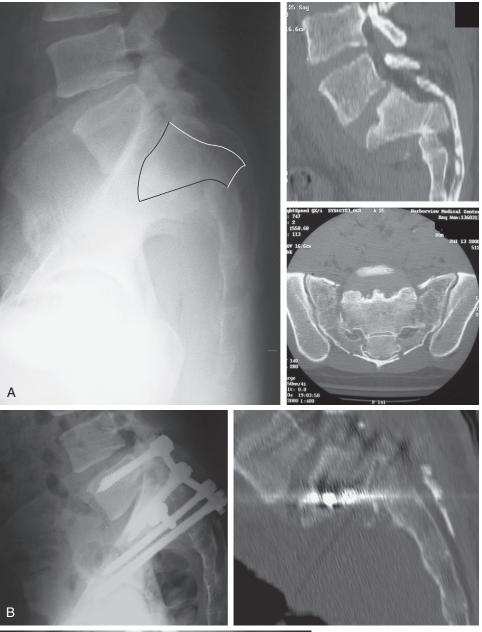
Fig. 40-12 A 19-year-old man who was crushed by a tractor and sustained a Roy-Camille type 2 sacral U fracture with incomplete cauda equina injury. *A, Clockwise from left,* Lateral radiograph, sagittal CT reformation, and axial CT view show comminuted bilateral transforaminal and transverse sacral fractures with severe kyphosis and canal compromise. Sacral roots were found to be contused but intact. *B,* Postoperative lateral radiograph and sagittal CT reformation, after L5 to S3 laminectomy and stabilization with bilateral iliosacral screws and segmental fixation from L5 to the ilium, show improved sacral kyphosis and decompression of the sacral canal. *C,* Postoperative AP radiograph of the pelvis showing the lumbopelvic fixation.
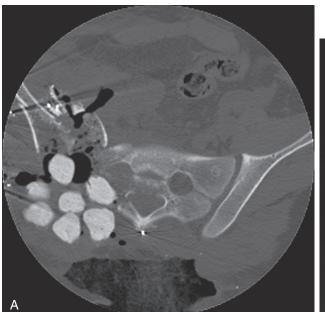
Fig. 40-13 *A,* Axial CT image of a 10-year-old girl who sustained a gunshot injury to the pelvis. She has previously undergone debridement and placement of antibiotic-impregnated methylmethacrylate beads. Note the comminution and bone loss at the right sacroiliac joint. *B,* AP radiograph of the pelvis 2.5 years after lumbopelvic fixation and right sacroiliac joint arthrodesis. (From Schildhauer et al: J Trauma 2007; 63:E95–99.)

requiring surgical stabilization, is much lower in the pediatric population.

Depending on their characteristics, sacral fractures in both adults and children can result in posterior pelvic ring instability, spinopelvic instability, or both. They are usually the result of high-energy injuries, although insufficiency and stress fractures may occur, particularly in adults. In both the adult and pediatric patient populations, vertical sacral fractures occur because of a combination of shear forces caused by oppositely directed forces from the lower extremity and the torso, rotational forces caused by lateral compression or external rotation of the iliac wings and flexion or extension movments across each hemipelvis. The same shear forces described above may cause bilateral–vertical sacral fractures associated with a transverse sacral fracture, which results in spinopelvic dissociation in addition to possible pelvic instability. Isolated sacral fractures occur from direct blows onto the posterior sacrum, and are generally located caudal to the sacroiliac joints where they do not influence pelvic ring or spinopelvic stability.

Patients with sacral fractures are often physiologically compromised and obtunded, making neurologic assessment difficult and incomplete. A careful assessment of the pelvic radiographs and CT images is essential to understand the severity of such injuries and to determine appropriate management. A physical examination should include the perineal region to assess for anal sphincter tone and the possibility of an open fracture. Although limited in scope, the literature suggests that neurologic injuries resulting from sacral frac-

tures usually improve regardless of surgical intervention. However, our experience is that surgical decompression and stabilization both facilitates and augments the degree of neurologic recovery. The relative likelihood of neurologic recovery in adults as compared with children is not known because of the lack of sufficient data. Appropriate stabilization methods are essential to maximizing functional outcome and involves iliosacral screw fixation, with or without ipsilateral lumbopelvic fixation for vertical sacral fractures and bilateral lumbopelvic fixation for fracture patterns that result in spinopelvic dissociation. The timing of surgery is a matter of debate, and must take into consideration the physiologic status of these often multi-injured patients. Given the uncertain relationship between the timing of decompression and the potential for neurologic recovery in patients with complex sacral fractures, emergent decompression is rarely indicated.

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C H A P T E R

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DANIEL H. KIM

Operative Techniques for Sacral Injuries: Surgical Approaches, Techniques for Decompression, and Instrumentation

INTRODUCTION

Sacral injuries, often undiagnosed and untreated, often result in neurologic symptoms; deficits to the lower extremities; and urinary, rectal, and sexual dysfunction. The neurologic problems often remain the major chronic sequelae after the more obvious pelvic trauma lesion has healed.[1] Treatment of injuries to the lower lumbar and sacral spine requires consideration of a number of additional factors beyond those relevant to injuries of the thoracic and thoracolumbar spine. These considerations are related to the anatomic complexity of the sacrum and the posterior elements of the lumbar spine and to the lordosis and improved mobility of the lumbosacral junction.[2]

Injuries to the lumbar spine and upper sacrum disrupt the normal lordotic alignment of the spine, and restoration of that lordotic alignment is critical to overall vertebral mechanics and spinal alignment in the sagittal plane. Failure to maintain or restore the normal sagittal alignment in the lower lumbar spine after either elective fusions or fractures can lead to degenerative changes and progressive symptoms during long-term follow-up. The lumbosacral junction in particular must resist a number of large forces but also must permit a significant amount of motion.[3]

The sacrum, with its intimate attachments to the pelvis, is a very stable structure. The relatively immobile sacroiliac joint and strong ventral and dorsal ligamentous attachments between the sacrum and pelvis account for the stability. The

sacrum lies at a 40-degree incline from horizontal at the lumbosacral junction. Axial loads, therefore, result in rotational forces. These rotational forces are resisted by the strength of the sacrotuberous and sacrospinous ligaments, which attach opposite the S4 neural foramina.[4] The sacrum forms a portion of the dorsal pelvic arch and, as a result, 90% of reported sacral fractures occur in conjunction with pelvic fractures.[5] Removal of the sacrum distal to the S1-S2 interspace weakens the pelvic ring by 30%, and resection up to S1, which removes half of the sacroiliac joint, weakens the pelvic ring by 50%.[6] Thus any discussion of stability of the sacrum must include a discussion of pelvic stability.[4]

Sacral spine fractures are classified by many systems. The classification allows the physician to determine the stability of the spine and, thus, to determine the need for conservative treatment, reduction, decompression, and type of stabilization. The system used plays a key role in determining whether operative or nonoperative treatment is initiated.

CLASSIFICATION OF SACRAL FRACTURES

DENIS CLASSIFICATION

The most comprehensive analysis of sacral fractures was reported by Denis et al.[1] in 1988 and consisted of 236 patients with sacral fractures in a series of 776 patients with pelvic injuries. The authors reported a 21% incidence of nerve injury associated with sacral fractures. To assist in analysis of the data, the authors performed anatomic cadaveric studies that showed the relationships among sacral nerve roots within the foramina.

The classification system is based on the direction, location, and level of sacral fractures. Three different vertical zones through the sacrum were identified as having characteristic clinical presentations (Fig. 41-1).[1] Zone 1 is the region of the ala. Zone 2 encompasses the region of the sacral foramina. Zone 3 encompasses the region of the central sacral canal. Injuries are assigned to the highest zone that the fracture line transgresses. Therefore, a fracture that passes through all three zones would be classified as a zone 3 injury.
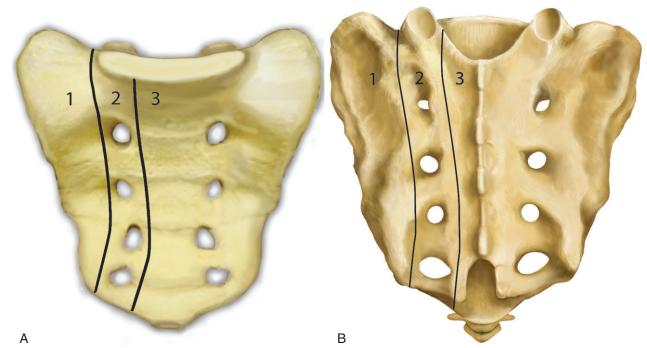
Fig. 41-1 Denis classification of sacral fractures. Zone 1 fracture is in the region of the sacral ala, zone 2 is through the sacral foramina, and zone 3 is through the central spinal canal. *A,* Anterior view. *B,* Posterior view.

Zone 1 fractures range from minor avulsion fractures to unstable vertical fractures lateral to the sacral foramina, with preservation of the sacroiliac ligaments. These injuries rarely present with neurologic deficit (5.9%) and occasionally are associated with partial damage to either the sciatic nerve or the L5 nerve root, thus presenting with symptoms of sciatica-type syndrome.[1] Vertical shear injuries of the pelvis can result in upward displacement of the sacral ala after a zone 1 injury. If severe enough, the upwardly displaced fragment can trap the L5 nerve root against the L5 transverse process, leading to a "traumatic far-out syndrome."[4,7]

Zone 2 fractures comprise fractures extending through one or more sacral foramina but lateral to the sacral canal. These fractures often occur in association with shear-type injuries but can also occur as the result of lateral compression. Vertical shear injuries are the result of significant force transmission to the sacrum and pelvis and usually result in concomitant injury to the sacroiliac joint.[4] Nerve root injury has been reported in 28.4% to 54% of zone 2 injuries.[1,8] Because these fractures usually are unilateral and do not involve the central canal, bowel and bladder injuries are not expected.

Zone 3 fractures, by definition, transgress the central canal and include vertical shear injuries, high and low transverse fractures, and traumatic spondylolisthesis of L5 on S1. Zone 3 fractures have a high incidence of bilateral nerve root injury and cauda equina dysfunction.[4] More than half (56.7%) of zone 3 fractures are associated with saddle anesthesia and bowel, bladder, and sexual dysfunction.[1]

ROY-CAMILLE CLASSIFICATION

Transverse fractures of the upper sacrum initially were classified into three groups by Roy-Camille et al.[9] The authors reviewed 13 cases of transverse sacral fractures and conducted cadaveric experiments to study the fracture mechanism. They noted that 11 of 13 cases resulted from suicide jumps, implicating axial loading as the predominant force of injury. Based on their clinical and biomechanical data, three distinct fracture types initially were described according to sagittal plane alignment[9] and were then modified by Strange-Vognsen and Lebech[10] to include a Type 4 (Fig. 41-2).

Type 1 fractures are flexion injuries with anterior simple bending of the upper fragment without significant translational displacement or deformity of the sacrum. Type 2 fractures are flexion injuries with posterior displacement of the cephalad fragment that becomes more or less horizontal and settles with its anterior surface on the fractured surface of the lower fragment. Type 3 fractures are extension fractures with anterior displacement of the cephalad fragment, more or less vertical, that slips down in front of the caudad fragment. Type 4 injuries are neutral position fractures with total comminution of the upper sacrum without displacement from the lower fragment.

CLASSIFICATION BY DIRECTION OF FRACTURE LINE

Fractures of the sacrum can be classified in a number of ways. One of the most common is according to the direction of the fracture line within the sacrum. Therefore, fractures can be
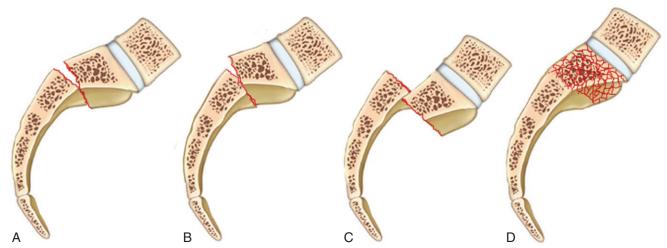
Fig. 41-2 Classification of transverse sacral fractures according to sagittal plane alignment by Roy-Camille et al. and Strange-Vognsen and Lebech. *A,* Type 1: flexion fracture with anterior simple bending of the cephalad fragment without significant displacement. *B,* Type 2: flexion fracture with posterior displacement of the cephalad fragment. *C,* Type 3: extension fracture with anterior displacement of the cephalad fragment. *D,* Type 4: neutral position fracture with total comminution of the upper sacrum without displacement from the lower fragment.

vertical, oblique, or transverse and can occur at any level in the sacrum (Fig. 41-3).[11]

Vertical fractures can occur in the ala or through the foramina. Oblique fractures can occur at any location. Transverse fractures are less common and more often occur at the apex of the sacral kyphosis between S2 and S3 but might occur as high transverse fractures at Sl or S2.[11]

The L5-S1 articulation can be disrupted by an oblique fracture of the sacrum. Extra-articular fracture of the base of the superior S1 facet can cause instability and spondylolisthesis at that level, or, alternatively, the fracture might directly disrupt the joint integrity.

MANAGEMENT OF LOWER LUMBAR AND SACRAL INJURIES

TREATMENT GOALS

The major goals of treatment for lower lumbar spine and sacral injuries are anatomic reduction of the injury deformity, rigid fixation, neural decompression (when indicated), maintenance of sagittal alignment, conservation of motion segments, and prevention of possible complications.[12] The time from injury must also be considered because the effectiveness of various methods changes over the course of time.

For most sacral fractures, treatment decisions are based on three factors of the classification schema: (1) it is important to know whether the sacral injury is isolated or accompanied by pelvic injury; (2) the fracture line must be identified as either vertical or transverse; and (3) if the fracture is oblique, it is important to ascertain whether it involves the L5-S1 articulation.[2]

The three principles for managing sacral fractures are as follows: (1) re-establishment of stability of the pelvic ring and lumbosacral junction, (2) correction and prevention of angular (kyphotic) and translational (shear) deformities of both the pelvic ring-sacrum and lumbosacral junctions, and (3) prevention of further neural deficit and treatment of existing neurologic injury with appropriate decompressive and stabilizing procedures.

TREATMENT PROTOCOL

The goals are a stable, pain-free pelvic ring and preservation of neural function.

CONSERVATIVE TREATMENT

Patients with intact neurologic function and minimally displaced or angulated fractures (e.g., stable zone 1 and 2 injuries) can receive conservative treatment. Care requires a short period of bedrest, then progressive mobilization in a cast or brace, and then ambulatory status as tolerated. During the treatment period, neurologic functions are monitored and fractures are assessed with serial radiographs. Because the site typically is well vascularized, the injuries are expected to be fully united by 3 months.

EXTERNAL FIXATOR

Unstable pelvic injuries are best treated with emergent application of external fixation. If vertical displacement is present, skeletal traction should be added.

SURGICAL TREATMENT

For vertical fractures, especially those occurring in zones 1 and 2 (with or without other pelvic involvement), fixation with horizontal compression across the posterior aspect of
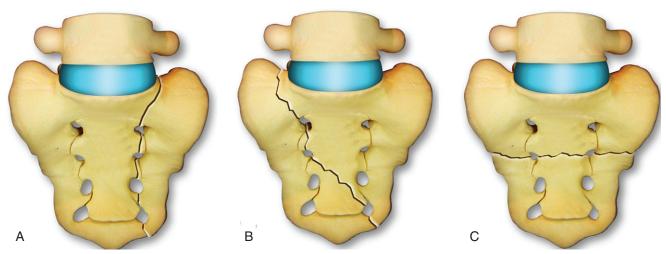
Fig. 41-3 Classification of sacral fractures by the direction of the fracture line. *A*, Vertical fracture. *B,* Oblique fracture. *C,* Transverse fracture.

the ilium and sacrum seems adequate. Although this can be accomplished by using sacral bars, the point of compression is posterior to the fracture line and the potential for an opening on the anterior portion of the sacrum exists unless anterior fixation is also used. In addition, for zone 2 injuries through the region of the foramen, compression is not desirable.

The sacral bar technique depends on compression for fixation and thus might compress the comminuted bone in the region of the foramen. Direct screw fixation can be applied with little or no compression. Recent biomechanical studies that evaluated fixation for transforaminal fractures showed little difference between one or two iliosacral screws in combination with posterior tension bands versus posterior transiliac bar devices.[2] Sacral bars have also been compared with a new plate devised for fixation of these fractures.

INDICATIONS FOR SURGICAL TREATMENT OF SACRAL INJURIES

1. Instability
   - Severe posterior ligamentous complex disruptions from a flexion or flexion-distraction injury
   - Gross ligamentous instability, such as bilateral facet dislocations with complete disruption of the posterior ligamentous complex and the disk, which will result in continued loss of sagittal alignment
   - Shear injuries with circumferential disruption
   - Burst injuries with significant canal compromise, disruption of the anterior and posterior portions of the vertebral body, and laminar fractures
   - Vertical sacral fractures with other pelvic ring fractures
   - Proximal transverse fractures of the sacrum with translational instability

2. Neurologic deficit
   - Direct neural decompression: a larger degree of canal compromise (50%) accompanied by high-grade neurologic compromise (cauda equina syndrome), specific root involvement with localized compression of the root, and patients with sagittal spinous process fractures, neurologic deficit, and dural tears with roots outside the dural sac
   - In the small group of patients with high transverse sacral fractures with kyphosis and neurologic deficit
   - Other neurologic injuries that accompany fractures of the sacrum are less likely to respond to direct operative intervention; a significant portion of these are root avulsions, and the remainder are neurapraxia, which often respond to conservative treatment
3. Disruption of axial or sagittal spinal alignment

APPROACHES TO LUMBOSACRAL JUNCTION AND SACRUM

The anterior approach is a transperitoneal or retroperitoneal approach used to expose the anterior aspect of the lumbosacral junction or sacrum. The posterior approach is used to expose the posterior aspect of the lumbosacral junction and sacrum. The bony, neural, and vascular anatomy of the region should be understood for both of these approaches. Additionally, by understanding the nature of the disease and the biomechanics of the lumbosacral pelvic area and spinopelvic fixations, surgeons will be able to better select the appropriate treatment for lumbosacral and sacral lesions.[13]

Lumbosacral iliac stability depends on the posterior bone and ligaments of the ilium and sacrum.[14] Sacroiliac stability is not greatly affected by resection if 50% or more of the sacroiliac joint is intact.[15]

SELECTION OF APPROACH TO LUMBOSACRAL JUNCTION AND SACRUM

The choice of approach to the lumbosacral junction and sacrum is determined by the site of the primary pathologic condition. Diseases involving the vertebral bodies can be approached directly through the abdomen or flank by using anterior or anterolateral approaches. Damage to the internal iliac vessels and presacral venous plexus, however, can be associated with significant hemorrhage and is a contraindication to anterior approaches to the sacrum. The posterior elements might be approached directly through a posterior incision in midline. The spinous processes, laminae, and facets are directly accessible through this approach, and the transverse processes and pedicles can be reached with somewhat more difficulty. The posterolateral approach provides direct access to the transverse processes and pedicles and limited exposure of the vertebral bodies themselves.

The anterolateral approach to the lumbosacral vertebral bodies through a long, oblique flank incision, beginning midway between the symphysis pubis and iliac crest, provides direct access to all the lower lumbar and upper sacral vertebral bodies in continuity for extensive resection or grafting at multiple levels. The incision is extended laterally and obliquely along the iliac crest to the midflank. A left paramedian longitudinal incision with retroperitoneal or transperitoneal dissections can also be used. The theoretical advantage of the transperitoneal approach is that the abdominal viscera might be more easily retracted than with a retroperitoneal dissection. The viscera and the hypogastric nerve plexus, however, are more vulnerable in this line of dissection from the front.

The posterior approach is the most common approach to the lumbosacral junction and the sacrum via a dorsal midline incision. The posterior midline approach is the most utilitarian and is most often used. The posterior approach through a posterior longitudinal incision in midline provides direct access to the spinous processes, laminae, and facets at all levels of the lumbar and sacral spine. The posterior aspect of the sacrum, the sacroiliac joints, the pedicles, and even transverse processes of the lower lumbar spine can be reached by retracting the paraspinal muscles laterally. This approach also provides adequate room for retraction of the cauda equina laterally to expose the ventral sacrum.[16] More extensive lateral exposure often is used to access the posterior aspect of the iliac wing and sacroiliac joints for placement of iliac screws or rods to stabilize concomitant lumbosacral and sacroiliac fracture-dislocations. In these cases, making an additional transverse T incision, located at the level of the iliac crests, can facilitate exposure.[17] Rarely, the most caudal extent of the midline approach can be used to access the coccyx for excision. The patient is placed in the prone position. It is important to keep the abdomen free from pressure, so that bleeding is minimized. The chest pad should be centered on the sternum, and the hip pads should be placed just distal to the anterior superior iliac spine to avoid undue pressure on the lateral femoral cutaneous nerve. Thighs should be well padded along the upper anterior portions to avoid pressure on the femoral nerve and postoperative palsy.

If a lumbosacral fusion is to be undertaken and supplemented with internal fixation, the lumbosacral junction should be placed in extension. This can easily be achieved by placing pillows or bolsters under the hips. In this position, the sacrum is prominent and constitutes the highest point on the table. The skin incision varies according to the pathologic condition and planned operation. A midline or paramedian vertical incision, transverse incision, upward arched incision, or downward arched incision can be used (Fig. 41-4).[13]

Fig. 41-4 Various skin incisions for posterior sacral approaches. *A,* Paramedian vertical incision for sacrospinalis-splitting approach. *B,* Transverse incision. *C,* Inverted-U incision.

Wiltse et al.[18] introduced an incision and retraction technique that uses one or two incisions 5 cm lateral to the midline and medial to the posterior superior iliac spine (Fig. 41-4, *A*). The dissection is deepened to the sacrospinalis muscle and the transverse process of the fifth lumbar vertebra. Wiltse et al. have used this approach for lumbosacral fusions. Bone grafts from the dorsal iliac crest can easily be obtained with the same exposure. The sacroiliac and sacrotuberous ligaments and the gluteus maximus muscle are divided as near to the sacrum as possible because their approximation before wound closure is necessary to avoid ventrodorsal postoperative wound complications.[13]

The paramedian posterior sacral approach is useful for open reduction and internal fixation of vertical sacral fractures that pass through or lateral to the sacral foramina (zone 2 or 1, respectively).[17] One advantage over the midline approach is that it allows more direct exposure of such fractures; however, it is less easily converted to an extensile approach of the lumbar spine. Direct open plating of vertical sacral fractures can be performed through the paramedian approach. Although open reduction of displaced transforaminal sacral fractures through this approach is critical to avoid iatrogenic nerve or vascular injuries, placement of iliosacral screws typically is performed percutaneously through separate lateral stab incisions through the gluteal muscles. A single transverse incision can also be used to expose the sacrum. It is most useful for reduction and fixation of transverse sacral fractures.[17]

The posterolateral approach through a longitudinal paraspinal incision, retracting the erector spinae muscles medially, provides direct access to the transverse processes and the facet joints. This approach is used for far-lateral disk herniation or for posterolateral fusion of the transverse processes. This area provides an excellent bed for posterolateral lumbosacral fusion. Through this approach, the transverse process can be removed and the pedicle and vertebral body can be exposed in a limited fashion. Wiltse et al.[18] explained that by using this approach, less muscle mass must be retracted medially, the facets are more directly reached, and operative hemorrhage might be less significant.[18]

OPERATIVE TECHNIQUES FOR SACRAL FRACTURES

ILIOSACRAL SCREW FIXATION

The technique of iliosacral screw fixation, in both prone and supine operative positions, has been well described.[11] The verification of reduction and screw placement can be done with either image intensification or computed tomographic guidance.

Whether the patient is in a prone or supine position, the operation must be performed on a radiolucent operating table. The image intensifier is placed on the side opposite the surgeon. Initial calibration of the angles is performed, which

is necessary to obtain simulated inlet (40 degrees caudad) and outlet (40 degrees cephalad) views for monitoring screw insertion (Fig. 41-5).

Anatomic reduction is performed by positioning or traction, and fluoroscopic confirmation is obtained. The position of the starting hole is the most critical feature for obtaining adequate screw position. A number of different techniques exist for determining the starting hole, but most are based on the intersection of two lines: one from the sciatic notch and one from the posterior superior iliac crest. Ideally, the screw should be perpendicular to the ilium and should cross the sacroiliac joint and remain within the sacral ala, proximal to the S1 foramen and distal to the L5-S1 disk, entering the vertebral body of S1.

The screw penetration must be at least to the midline for zone 1 fractures and across the midline for zone 2 injuries. Penetration should cross the midline into the contralateral ala for zone 3 fractures with an oblique or vertical component.

It is preferable to use a 6.5-mm fully threaded cannulated cancellous screw. This prevents further compression across the comminuted fracture site, which decreases the chance for impingement of roots in transforaminal fractures. Screw length generally averages between 60 and 90 mm, but as much as 130 mm might be required to reach the contralateral ala. Use of the calibrated direct measurement on the image intensifier makes choice of screw length easier. Washers should be used to prevent penetration of the screw head into the ilium.[2]

BILATERAL SACRAL PLATE OR SCREW FIXATION

The technique of bilateral plating or screw fixation of the posterior surface of the sacrum is used for patients with isolated oblique or transverse fractures of the sacrum, with or without involvement of the lumbosacral junction.[11] It can also be used for fractures that are unstable and have significant deformity and for patients with neurologic deficit.

The patient is placed in a prone position on a radiolucent operating table, with the table slightly flexed at the midportion. The exposure should be a wide exposure of the spine from L5 down to S4 through the midline (Fig. 41-6).

During posterior dissection in the area of the fracture, care must be taken to avoid motion of the comminuted fragments to prevent further damage of the compromised nerve roots. The dissection is made laterally beyond the dorsal foramen at all levels from S1 to S4. The L5-S1 facet joint capsules are preserved if no involvement is present at L5-S1. Otherwise, the L5-S1 facet joint capsules can be removed. The posterior aspect of the fracture line can be fully identified after exposure of the posterior aspect of the sacrum. Placement of the plate generally is in a line directly over the dorsal foramina.

A sacral laminectomy is performed to identify the sacral nerve roots and the area of maximal deformity and compression. The decompression must extend laterally enough to

Anteroposterior

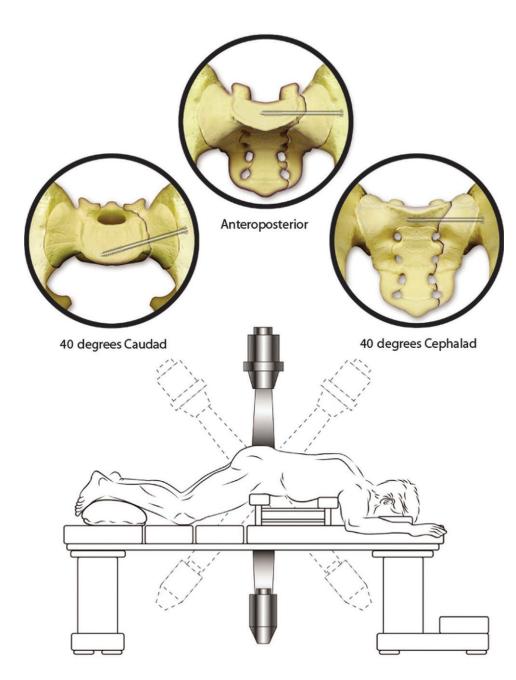40 degrees Caudad

40 degrees Cephalad

Fig. 41-5 Iliosacral screw fixation. To be certain that iliosacral screw fixation into the sacrum is accurate, some type of monitoring is necessary. The image must be placed so that views 40 degrees caudad and 40 degrees cephalad can be obtained during the procedure.

identify the take-off point of the ventral root sleeves and the bone of the vestigial sacral pedicles. Once the deformity is identified and the laminectomy is completed to enable direct visualization of the roots, attention is turned to reduction of the fracture. Little attempt is made at this time to decompress the roots, as reduction of the kyphosis often accomplishes much of this. Care should be taken, however, to ascertain that the ventral surface of the roots at the level of the maximal fracture angulation is not trapped in the fracture site.

Because the sacral roots are freely visible after the decompression, a Cobb elevator can be safely placed within the fracture to lever the fragments or bone clamps can be used

to apply corrective translational forces to reduce the fracture.

Intraoperative use of the image intensifier often is helpful at this stage. Complex multiplanar fractures can be reduced in a similar fashion, with the possible addition of intraoperative skeletal traction to reduce vertical shear.

For fractures that are predominantly transverse, the fracture line can be identified in the region of S2-S3 and the dissection continued laterally to allow complete exposure of that fracture line. The fracture line and obliquity of the fracture line are identified with the use of a small curette, with care taken to remove as little as possible of the crushed cancellous bone during the procedure.
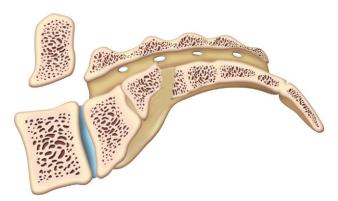
Fig. 41-6 Lateral view of the transverse sacral fracture with kyphotic deformity. A wide exposure from L5 to S4 is required for bilateral plating of the posterior sacrum.

Cobb elevators can then be placed within the fracture on both sides so that the kyphosis of the fracture can be corrected manually. The direction of application of leverage of the fracture line is based on the direction of displacement. If the proximal fragment is lying posterior to the distal fragment, then an attempt is made to pass the Cobb elevators over the superoanterior aspect of the distal fragment, after which the proximal fragment is gently levered into a less kyphotic configuration and is translated anteriorly.

If reduction can then be maintained by using a single Cobb elevator, instrumentation can be initiated. For more oblique fracture lines that might pass through the central sacral canal from a 20- to 45-degree angle, some foreshortening of the sacrum might occur. Length can be regained temporarily by placing bicortical screws through the sacral ala and then using a pelvic reduction clamp to slightly distract the screws to disimpact the fracture line and achieve length for correction of angulatory deformity.

If a large fragment of the floor of the sacral canal continues to compress the dural sac, even after initial temporary reduction, consideration is given to the excavation of the sacral ala just lateral to the canal so that the fragments that are impinging on the canal can be removed through an oblique lateral exposure. This might require bilateral excavations at the level of the fracture or simply unilateral excavation.

No attempt should be made to tamp the fragments into place. Rather, the fragments should be removed with the use of a pituitary rongeur and put aside for subsequent bone grafting. Care is taken not to damage the nerve roots during removal of the fragments.

At this point, internal fixation can be accomplished. Commonly, pelvic reconstruction plates are used to achieve stabilization. This might require either a 3.5- or 4.5-mm plate. The characteristics on which the choice of this hardware is based are its malleability over the posterior contours of the sacrum and the correlation of hole spacing with the proper fixation points on the sacrum.

For transverse or transverse oblique fractures within the body of the sacrum, a minimum of two sets of screws proximal and distal to the fracture line is preferable (Fig. 41-7). Because bone stock and screw fixation points are limited in the sacrum, reduction should not be attempted using the instrumentation to avoid screw pullout while levering on the plate. If the fracture configuration permits, insertion of both medial and lateral screws into the sacrum at S1 is preferred, thereby achieving optimal fixation into the superior fragment.

The most proximal screw, at the lateral border of the S1 superior facet, is directed approximately 30 degrees medially into the S1 body through the pedicle, aiming at the sacral promontory. The next screw, at the inferior edge of the S1 facet, is directed 35 degrees laterally into the sacral ala and parallel to the sacral endplate. In the remainder of the levels, a single or double screw traversing the pedicle laterally and parallel to the sacroiliac joint is preferred.

The average proximal screw inserted at the level of the medial border of the S1 facet usually is directed approximately 30 degrees medially, entering into the S1 body. The next screw is inserted below the inferior edge of the S1 facet and is directed laterally approximately 35 degrees into the sacral ala and parallel into the plane of the sacroiliac joint. The proximal screws generally average approximately 30 to 45 mm in length. The screws at S2, S3, and S4 are shorter (approximately 20 mm at the most distal point). Trajectory is between 20 and 35 degrees laterally.
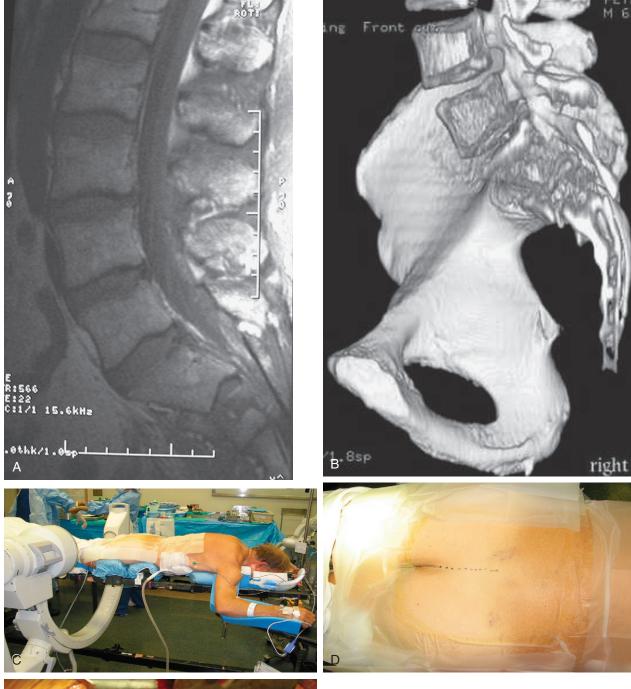
If the fracture line is more oblique than transverse and involves the L5-S1 articulation, the most proximal end of the construct must be extended to the L5 pedicle. This is sufficient if minimal translation is less than 20% of body width. However, the construct might need to be extended even more proximally to L4 to achieve adequate purchase on the distal portion of the spine if 50% or more displacement is present at the L5-S1 level.

After fixation is completed, the canal is reassessed. The decompression of the sacral nerve roots is reconfirmed by gentle retraction of the roots toward the midline. If any residual impinging bone remains, it is removed with a curette or pituitary rongeur. Tamping the fragments down into the sacrum is much less effective.

The contralateral side is also checked, and if reduction was incomplete, decompression is still possible by resecting the residual bone of the displaced portion of the floor of the canal. Decompression should not be attempted without stabilization, because shifting of the fracture alignment can easily cause compression to recur.

If reduction is incomplete and some translational deformity still exists but the fracture is in a stable position, additional excavation of the floor of the canal will be undertaken to remove any bone that might be pressing on the ventral surface of the sacral roots.

Cancellous grafting usually is not necessary for fractures contained totally within the sacrum, although any residual
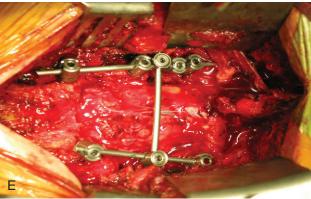
Fig. 41-7 Type 2 transverse sacral fracture treated with bilateral sacral screw fixation. *A,* Preoperative sagittal T1-weighted magnetic resonance image. *B,* Three-dimensional reconstructed lateral view. *C,* Position of the patient and image intensifier. *D,* Midline skin incision. *E,* Intraoperative photograph.

Continued

Fig. 41-7, *F,* Postoperative anteroposterior and lateral view radiographs.

graft can be used to fill in the defects. For those that traverse the L5-S1 articulation, however, standard transverse process and sacral alar grafting should be performed with autologous cancellous bone. Reapproximation of the paraspinous musculature over the hardware and fracture is critical.

Postoperatively, the patient is placed into a custom-molded total-contact orthosis, with one thigh incorporated into the brace. Once in the brace, the patient progresses to ambulation as tolerated. Bracing typically is continued on a full-time basis for 10 to 12 weeks.

PEDICLE SCREW AND ILIAC FIXATION WITH FOUR-SCREW FOUNDATION

The technique of iliac fixation with four-screw foundation can be used for patients with isolated oblique or vertical fractures of the sacrum, with or without involvement of the lumbosacral junction (Fig. 41-8).[2,19] Various systems with top loading polyaxial pedicle screws and iliac bolt screws can be used for stabilization of the lumbosacral and sacroiliac junctions. The Isola Spinopelvic System (De-Puy Acromed, Rayham, MA) incorporates traditional Isola designs with a new class of fixation options for a simplified connection to the pelvis, with specific emphasis on deformity, sacral trauma, and tumor applications.[19]

Proximal **anchor site preparation for lumbar and sacral pedicle screws**: Lumbar and sacral pedicles are prepared with awls, pedicle probes, ball-tip feelers, and bone taps. Appropriate insertion of lumbar and S1 pedicle screws is completed (Fig. 41-9, *A*).

Distal **anchor site preparation for superior and inferior iliac screws**: The iliac screw entry points are identified and prepared by resecting the posterior superior iliac spine with a curved osteotome. The resection should be flush with the sacrum to prevent entry into the sacroiliac joint. This also will place the iliac foundation more
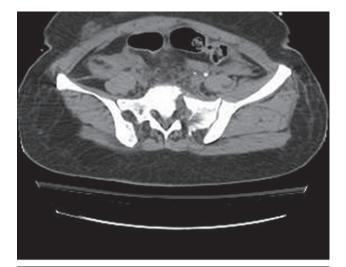

Fig. 41-8 Bilateral zone I vertical fractures of the sacrum managed with pedicle screw and iliac fixation. *A,* Preoperative axial view computed tomographic scan.
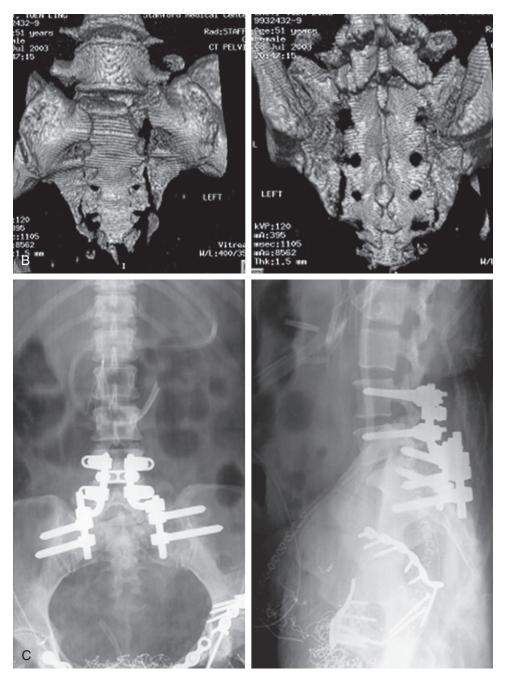
Continued

Fig. 41-8, *B,* Three-dimensional reconstructed anterior and posterior views show bilateral vertical fractures in the sacrum. *C,* Postoperative anteroposterior and lateral view radiographs.

anteriorly, improving the soft tissue coverage over the ilium. The resection results in a flat and oval-shaped cancellous starting point for both the superior and inferior iliac screws (Fig. 41-9, *B*).

Inferior iliac screw: With the fingertip of the opposite hand in the sciatic notch, the Isola iliac starter probe is inserted into the inferior portion of the oval cancellous window and is worked between the two cortical plates of the ilium. The standard Isola iliac probe is then inserted and advanced to just above the sciatic notch. The surgeon should palpate the lateral wall of the ilium when advancing the

Isola iliac probe. When the desired depth is reached, an anteroposterior view plain radiograph can be obtained with the probe in place. Fluoroscopy can be used at the discretion of the surgeon (Fig. 41-10, *A*). A ball-tip feeler is inserted into the prepared channel to confirm that the path has not perforated the pelvis (Fig. 41-10, *B*).

The size of the inferior iliac screw is determined. The recommended inferior screw is a large-diameter, long-length screw, such as a 10-mm Isola closed iliac screw. The large diameter of the screw is intended to purchase both iliac cortices for maximum fixation. The average length is
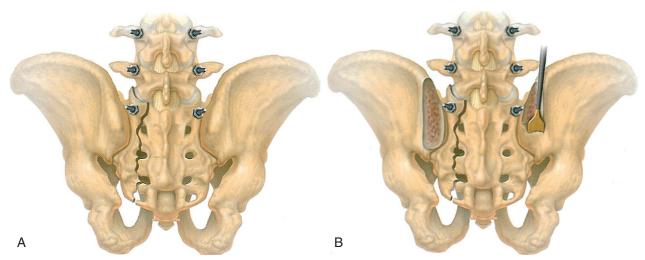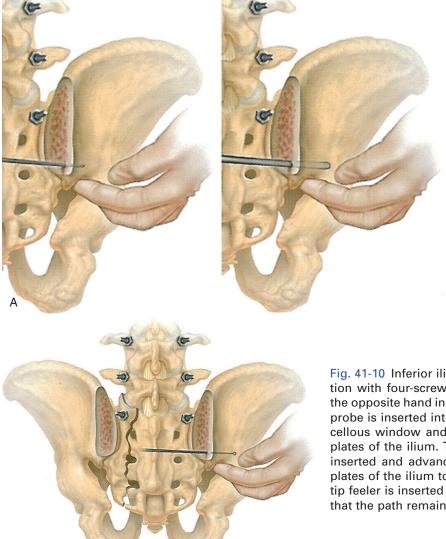
Fig. 41-9 Iliac fixation with four-screw foundation. *A,* Proximal anchor site preparation and proper insertion of lumbar and S1 pedicle screws. *B,* Distal anchor site preparation with a curved osteotome.

Fig. 41-10 Inferior iliac screw site preparation of iliac fixation with four-screw foundation. *A,* With the fingertip of the opposite hand in the sciatic notch, the Isola iliac starter probe is inserted into the inferior portion of the oval cancellous window and is worked between the two cortical plates of the ilium. The standard Isola iliac probe is then inserted and advanced further between the two cortical plates of the ilium to just above the sciatic notch. *B,* Ball-tip feeler is inserted into the prepared channel to confirm that the path remains between the iliac cortices.

70 mm, but some patients will accept up to a 100-mm screw (Fig. 41-11, *A*). The inferior iliac screw path is tapped, and a closed Isola iliac screw is inserted into the inferior ilium (Fig. 41-11, *B*). Some key points to remember include the following: (1) the iliac screw should be placed as perpendicular as possible to the coronal plane of the spine, (2) the inferior screw should not be inserted at an angle like a traditional Galveston position, (3) screw position is critical for ease of construct assembly, (4) minimal convergence of the iliac screws is optimal because it allows the contoured rod to pass easily through the screw heads, and (5) the opening of the screw should face cephalad and caudad.

Superior iliac screw: The insertion site of the second iliac screw is the superior end of the oval cancellous window. The Isola iliac starter probe is inserted into the superior end of the oval cancellous window with a direction of slight cephalad inclination. The standard Isola iliac probe is then inserted to widen the path in the ilium. The surgeon palpates the outer upper ilium to help guide probe placement. It is recommended that the probe be inserted to a depth of 45 to 55 mm (Fig. 41-12, *A*).

The superior iliac screw path is tapped, and a closed Isola iliac screw is inserted into the superior ilium. The opening of the superior iliac screw faces cephalad to caudad
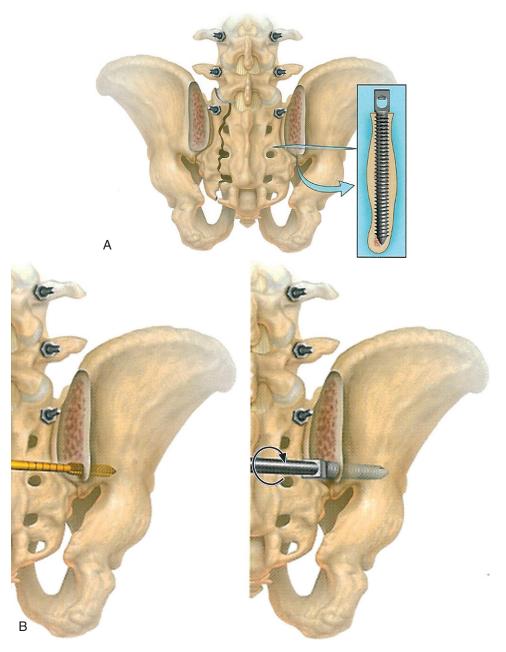

Fig. 41-11 Inferior iliac screw selection *(A)* and insertion *(B)*. Large-diameter (10 mm), long-length screw is recommended for the inferior iliac screw.
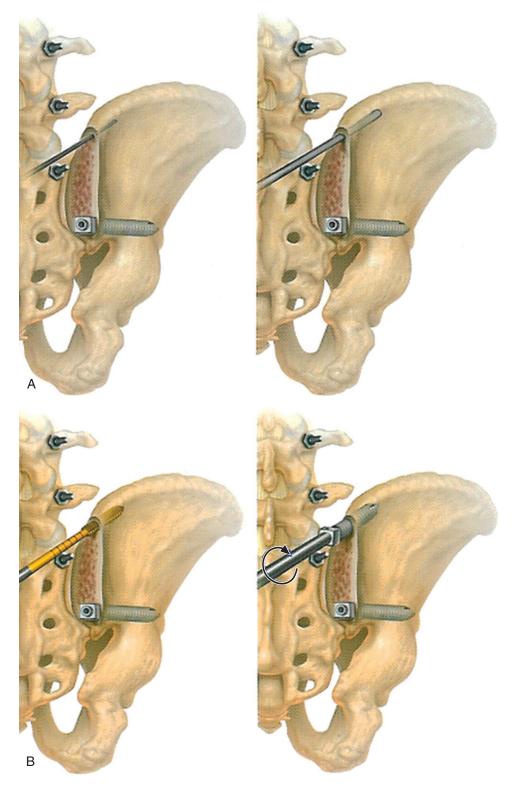
Fig. 41-12 Superior iliac screw site preparation and screw insertion. *A,* Second iliac screw insertion site is identified at the superior end of the oval cancellous window. Isola iliac starter probe is inserted into the superior end of the oval cancellous window with a slight cephalad inclination. Standard Isola iliac probe is then inserted to widen the path in the ilium. *B,* Superior iliac screw path is tapped and a closed Isola iliac screw inserted into the superior ilium. Superior iliac screw is positioned with the opening of the screw facing cephalad to caudad.

(Fig. 41-12, *B*). The recommended insertion depth of the superior iliac screw is 45 to 55 mm. A 10-mm screw can be used in most patients; however, a 7.75-mm screw can also be used. The screw is inserted with a slight cephalad inclination and will not be perpendicular to the resection site as seen with the inferior screw.

Sizing and trial of distal anchor rod: The distal anchor site preparation procedure is repeated for the opposite side of the pelvis. Rods are measured, cut, contoured, and provisionally placed through the openings of the closed screws to verify fit (Fig. 41-13). Rods are then removed, and rodded connectors are added. The assemblies are

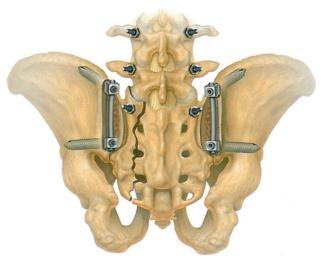Fig. 41-13 Distal anchor site preparation procedure is repeated for the opposite side of the pelvis. Rods are measured, cut, contoured, and provisionally placed through the openings of the closed screws to verify fit.

placed into the closed screws. Unobstructed passage of the rod through the screws should be confirmed.

Interconnection of proximal and distal anchors: The proximal and distal anchors are interconnected with the use of 6.35-mm rodded connectors and standard Isola slotted connectors. This creates independent proximal and distal foundations from which the ilium and sacrum can be manipulated to achieve correction. All nuts on the pedicle screws are provisionally tightened to provide stability to the construct during corrective maneuvers (Fig. 41-14, *A*). Four rodded connectors are required to interconnect the lumbar, sacral, and iliac anchors to one another. The first pair of rodded connectors (cephalad to caudad), running from the ilium into the sacrum and up into the lumbar region, is used to correct the displaced ilium and sacrum. The second pair of rodded connectors (medial to lateral), spanning across the sacrum, is used to reduce the fracture.

Proximal foundation manipulation and correction of displaced sacrum and ilium: With the use of distraction between L5 and S1, the displaced sacrum and ilium are brought back into proper anatomic alignment. Set screws are locked.

Distal foundation manipulation and reduction of fracture: The fracture is reduced with the use of compression between the transverse rodded connector and the side-by-side dual rod connector. Set screws are locked.

Final tightening: The set screws on the slotted or rodded connectors and closed screws are tightened to 60-inch pounds, and then final tightening is performed with the hex nut on the pedicle screws to 100-inch pounds. A cross connector is added at the desired level (Fig. 41-14, *B*).

Biomechanical study shows the effectiveness of adding a second fixation point distal to the S1 screws in reducing S1 screw strain.[20] Iliac fixation is more effective than the use of secondary sacral fixation points but might not be necessary in all clinical situations.[20] A technique incorporating independent proximal and distal foundations, composed of lumbar and sacral pedicle screws and iliac screws, can facilitate reduction maneuvers required to restore pelvic stability and balance to promote solid fusion (Fig. 41-15).[19,21]

OTHER TECHNIQUES FOR TREATMENT OF SACRAL FRACTURES

Other variable techniques are available. Sacroiliac plate, sacral bar, tension band across the ilium, and transiliac screw techniques and their combinations have been developed. Some of the techniques are shown in Figure 41-16.

A   B
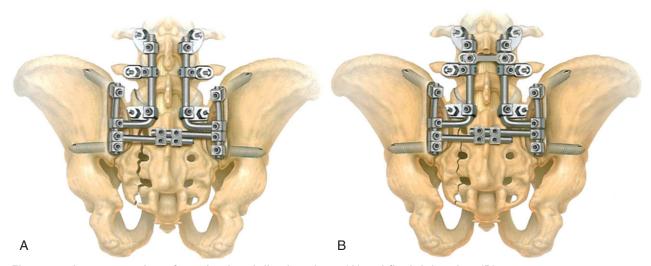
Fig. 41-14 Interconnection of proximal and distal anchors *(A)* and final tightening *(B)*.
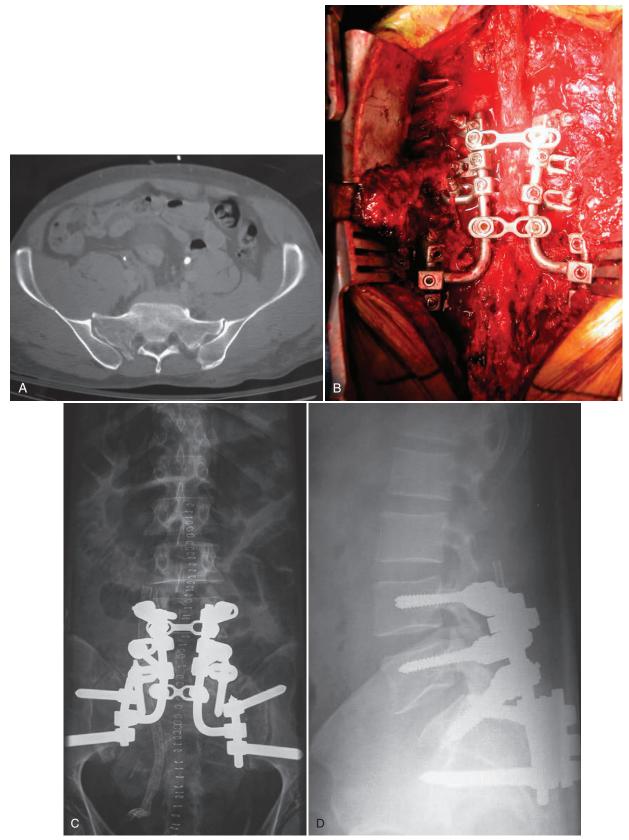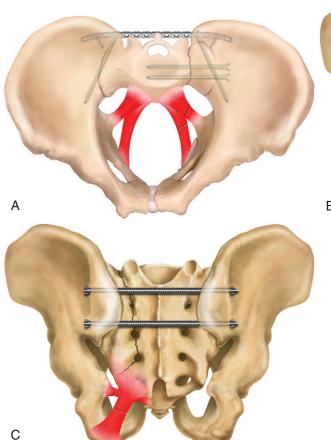
Fig. 41-15 Case of zone 1 vertical fracture of the sacrum was managed with pedicle and iliac screw fixation with a four-screw foundation. *A,* Preoperative axial view computed tomographic scan. *B,* Intraoperative photograph of pedicle and iliac screw fixation. *C,* Postoperative anteroposterior view radiograph. *D,* Postoperative lateral view radiograph.
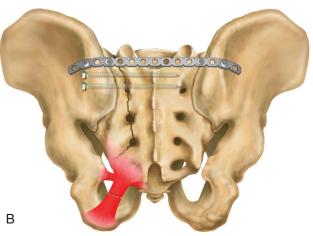
Fig. 41-16 Various techniques for application of iliosacral screws plus tension band plate across the ilium (*A* and *B*) and transiliac screws *(C)* for the treatment of sacral fractures.

COMPLICATIONS ASSOCIATED WITH OPERATIVE TREATMENT FOR SACRAL FRACTURES

INTRAOPERATIVE COMPLICATIONS

Complications associated with posterior approaches for the lower lumbar and sacral fractures might include dural tear, neurologic deficits or deterioration, and vascular injury.[17,22,23] Dorsal rami injury is an expected complication in association with sacral exposure beyond the foramina. Sacral root injury can occur with associated procedures such as pedicle screw insertion, iliosacral screw placement, or decompressive laminectomy.[17] Superior gluteal artery injury can occur with overly aggressive exposure of the lateral sacrum. This can lead to intraoperative massive hemorrhage. Because the transected artery usually retracts into the pelvis, an emergent anterior pelvic exploration usually is necessary to gain control of the hemorrhage. Superior and inferior gluteal nerve injury can denervate the gluteus muscles, leading to ambulation difficulty.

Specific complications that can occur in association with anterior transperitoneal or retroperitoneal surgery to the lumbosacral junction include arterial thrombosis, venous thrombosis, various injuries to the great vessels, urogenital structures (ureter and bladder), intra-abdominal viscera, and neurologic elements (cauda equina, sympathetic and parasympathetic plexus) and the formation of abdominal wall hernias.[24] Vascular injury can be one of the most devastating injuries to occur during surgery of the anterior lumbosacral junction. A vascular surgeon should be available in the event of such an injury. The surgeon should be especially cautious when working on the lower lumbar spine because the left common iliac vein crosses the L4-L5 disk space and commonly is injured.[25] Intraoperative vascular injury during anterior lumbar surgery is not infrequent and was as high as 16% in one series.[26] The sympathetic trunk can be injured during dissection of the psoas.[25] The injury will manifest itself as an ipsilateral warm extremity and usually is not a significant clinical injury. In the lower lumbar spine, the superior hypogastric plexus can also be injured. This injury is of special concern in male patients because it can lead to retrograde ejaculation. The reported incidences of retrograde ejaculation vary in the literature from 0.42% to 5.9%.[27,28] Male patients should be counseled preoperatively regarding this possible complication.

POSTOPERATIVE COMPLICATIONS

Possible postoperative complications associated with surgical treatment of lower lumbar and sacral fractures include thromboembolism, deep wound infections, pseudarthrosis, implant failure requiring removal or revision, and loss of correction.[17,22,23] Wound infection more often occurs in patients who have sustained high-energy fractures associated with extensive soft tissue injuries.[17] Deep wound infection with persistent pustular drainage lasting more than 7 to 10 days after surgery should be treated with open surgical debridement and irrigation in the operating room.

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Fractures of the Sacrum and Coccyx

INTRODUCTION

Although sacral fractures can occur in isolation or in low energy traumas, they are most commonly associated with pelvic-ring injuries. An accurate estimate of fracture incidence is difficult to make because missed and delayed diagnoses are not uncommon; the practitioner should, therefore, have a high index of suspicion in the appropriate clinical context. Because these injuries most often occur as a result of high energy trauma, patients should be carefully assessed for other orthopaedic and nonorthopaedic injuries with a complete physical examination and appropriate imaging studies. Treatment options range from nonoperative management to surgical neural decompression and fracture stabilization. Detailed neurologic and radiologic examination (often computed tomography) along with consideration of the patient's overall status help guide the medical decision-making process. Timely evaluation and treatment are important because neurologic deterioration has been associated with radiologic evidence of neural compression. However, reports of long-term outcomes of surgical and nonsurgical treatments are limited. In contrast, fractures of the coccyx usually occur in isolation and result from a direct impact to the central buttock region with the hips in a flexed position. These fractures are typically managed symptomatically with surgery indicated in cases of significant coccydynia.

EPIDEMIOLOGY AND PRESENTATION OF SACRAL FRACTURES

A small minority of sacral fractures occurs in isolation (less than 5%), most commonly as a result of a direct impact to the area.[1] Because of the importance of the sacrum and associated ligamentous structures to posterior pelvic stabil-

ity (Fig. 42-1), the majority of sacral fractures evaluated are concomitant with pelvic fractures—in fact, 45% of patients with pelvic fractures also have a sacral fracture—and occur in the context of a high energy trauma: 57% result from motor vehicle crashes, 9% from motorcycle crashes, 18% from pedestrian–motor vehicle injuries, 9% from falls, and 4% from crush injuries.[2,3] Certain mechanisms of injury should raise increased suspicion for particular fractures patterns: a falling injury is more likely to show a transverse pattern (direct posterior to anterior impact) and a pedestrian–motor vehicle impact is more likely to show a lateral compression pattern (indirect impact through the ilium).

Because patients with these fractures have multiple, often more prominent injuries, many sacral fractures are missed on initial presentation; indeed, up to 30% are found as a late diagnosis.[4] Therefore, a high index of suspicion must be maintained if there is a history or other evidence consistent with significant direct or indirect pelvic trauma. An undiagnosed sacral fracture may lead to progressive pelvic instability leading to a nerve root compression and potentially adverse outcomes. Indeed, sacral fractures have been shown to have a high rate of neurologic injury (25%), a finding consistent with the intimate nature between the bony sacrum and the lumbosacral plexus.[2]

The large retrospective study (though limited because of sampling bias) showed the prevalence of fractures of the sacrum to be 51% in patients presenting without a neurologic deficit and 76% in patients presenting with a deficit.[2] The risk for neurologic injury was stratified using the following classification scheme (Fig. 42-2, A). Zone 1 fractures (lateral to neural foramina and the most common fracture pattern at 50%) had the lowest prevalence of neurologic impairment, with 6% involving the L5 nerve root or sciatic nerve (Fig. 42-2, B). Zone 2 fractures (disruptions through the neural foramina and the second most prevalent pattern at 34%) were associated with L5, S1, S2 nerve root injuries 28% of the time. Finally, zone 3 fractures (disruptions central to the foramina) had the highest risk of neurologic injury (60%)—of these patients, 76% experienced bowel, bladder, and sexual dysfunction. In addition, transverse fracture patterns involving S1 to S3 have been associated with higher
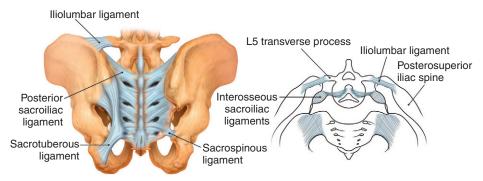
Fig. 42-1 The relationship between the sacrum and the major posterior stabilizing ligamentous structures of the pelvic ring. (Adapted from Tile M, Helfet DL, Kellam JF: Fractures of the Pelvis and Acetabulum, 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2003.)
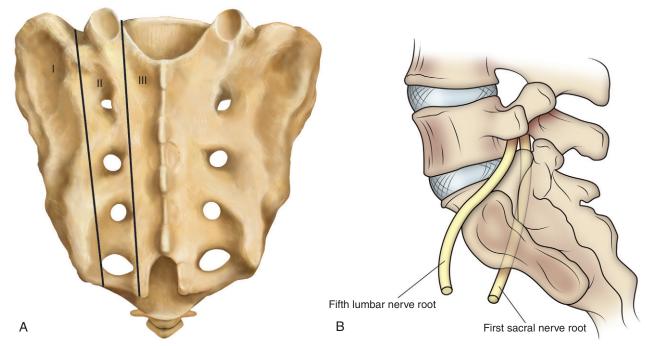
Fig. 42-2 A, Zone 1 fractures are lateral to neural foramina, the most common fracture pattern at 50% and have the lowest prevalence of neurologic impairment at 6% involving the L5 nerve root or sciatic nerve. Zone 2 fractures are disruptions through the neural foramina, the second most frequent pattern at 34% and are associated with L5, S1, S2 nerve root injuries 28% of the time. Zone 3 fractures are disruptions central to the foramina and have the highest risk of neurologic injury at 60%. B, The relationship between the sacrum and lumbar and sacral nerve roots are shown. (Adapted from Routt MLC Jr, Simonian PT, Agnew SG, Mann FA: Radiographic recognition of the sacral alar slope for optimal placement of iliosacral screws: A cadaveric and clinical study. J Orthop Trauma 10:171, 1996.)

rates of bladder dysfunction when compared with those involving the S4 nerve roots and below.[3]

INITIAL EVALUATION OF SACRAL FRACTURES

A thorough evaluation must be performed on any patient with a suspected sacral fracture. Initially, a systematic examination in accordance with the Advanced Trauma Life Support (ATLS) protocol should be performed as for any patient with evidence of significant high-energy blunt trauma.[3] For the orthopaedist, the presence of lacerations, contusions, or other evidence of significant soft tissue injury in the pelvic area should raise suspicion for a sacral fracture; with concomitant fluctuance or crepitance, there may be a fascial degloving (Morel-Lavallee lesion) a finding that could limit options for fracture fixation.[5,6] There are data to suggest that these lesions may be treated with early percutaneous drainage and debridement or with the more traditional open debridement and packing.[6]

For patients with known sacral fractures, lumbosacral neurologic examination including testing of motor and sensory function and deep tendon reflexes must be executed. Along with a peripheral neurologic examination, a more critical, axial evaluation via a manual rectal examination that evaluates concentric perianal sensation (S2 to S5 distribution), spontaneous and voluntary sphincter tone, and reflexes (perianal wink, bulbocavernosus, and cremasteric) is essential.[3] In the presence of a neurologic deficit, assessment of the vascular status of the lower extremities should be documented. In addition to absent femoral, popliteal or distal pulses, a low ankle-brachial index suggests vascular injury and may indicate the need for angiography. Bladder dysfunction present with sacral fractures can be due to neurologic compromise, direct damage from fracture fragments, or indirect trauma from the initial blunt impact. A basic workup (serum blood urea nitrogen [BUN]/creatinine concentrations, ultrasound or urodynamic evaluation) should be initiated and urologic consultation obtained. For female patients, a vaginal exam should be performed to assess for a hidden open fracture. In addition, patients should be maintained on full spinal precautions until spinal evaluation is complete because noncontiguous thoracolumbar fractures are not uncommon and should be stabilized first.[7]

FRACTURES OF THE COCCYX

The epidemiology of coccygeal fractures is not well known. These fractures are usually the result of low to moderate energy injury, do not require surgical intervention, and are seldom reported in the medical literature. The prevailing mechanism is a direct impact to the coccyx from a backward fall onto a seated position. The two groups at greatest risk for these fractures are participants in athletic activity and those patients already at risk for fragility fractures (older age, female, presence of osteoporosis or other metabolic bone disease). Other, more unusual cases during childbirth or soccer celebrations have also been reported.[8,9] Patients may present to an emergency or family physician with a history of a fall and pain most prominent with sitting—only those with significant or persistent discomfort will come to the attention of an orthopaedic surgeon. If the fracture is greatly displaced, the result of a high-energy impact or associated with other significant injuries, a more thorough trauma-oriented evaluation including a manual rectal examination and careful documentation of neurologic status is warranted. Neurologic compromise is extremely rare with isolated coccyx fractures and should alert the practitioner that other injuries may be present.[10]

CLINICAL MANAGEMENT

RADIOLOGIC EVALUATION

Radiographic evaluation of potential sacral fractures should be performed within the context of the larger trauma workup; initial imaging should include the following standard radiographic views: anteroposterior chest, lateral cervical spine, and anteroposterior pelvis. Signs on the anteroposterior view suggestive of sacral fracture include L5 (and to a lesser extent L4) transverse process fractures, anterior pelvic ring disruptions, and a "stepladder" sign (appearance of overriding transverse fracture fragments as a result of anterior sacral foraminal and lumbosacral facet disruption).[11] However, because of the anatomic inclination of the sacrum relative to the coronal plane of the pelvis (Fig. 42-3, A), anteroposterior radiographs alone fail to detect the majority of sacral fractures; supplementation with inlet and outlet images can increase sensitivity.[12] The lateral view of the sacrum can also be useful for detecting presence of injury but rarely provides useful bony detail (Fig. 42-3, B).[3,12,13]

The imaging modality of choice for more detailed bony evaluation of the sacrum and coccyx remains computed tomography (CT) performed with fine sectioning (1–2 mm) and multiplanar (coronal and sagittal) reconstruction views (Figs. 42-3, C to E).[2,14] For more specific assessment of potential neural compression, magnetic resonance imaging (MRI) is the modality of choice over CT myelography because the thecal sac can terminate at the S1-S2 junction. [3] MRI has the added advantage of being noninvasive and providing additional diagnostic information regarding the remainder of the pelvis, spine, or both.

TREATMENT RATIONALE FOR SACRAL FRACTURES

Because sacral fractures can herald a complex set of injuries systemically and to other organ systems in and around the pelvis, the decision-making process and timing of treatment must be made within the context of the overall condition of the patient; the orthopaedic practitioner must do so in
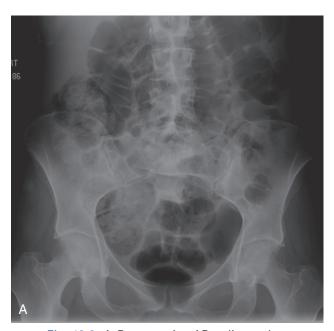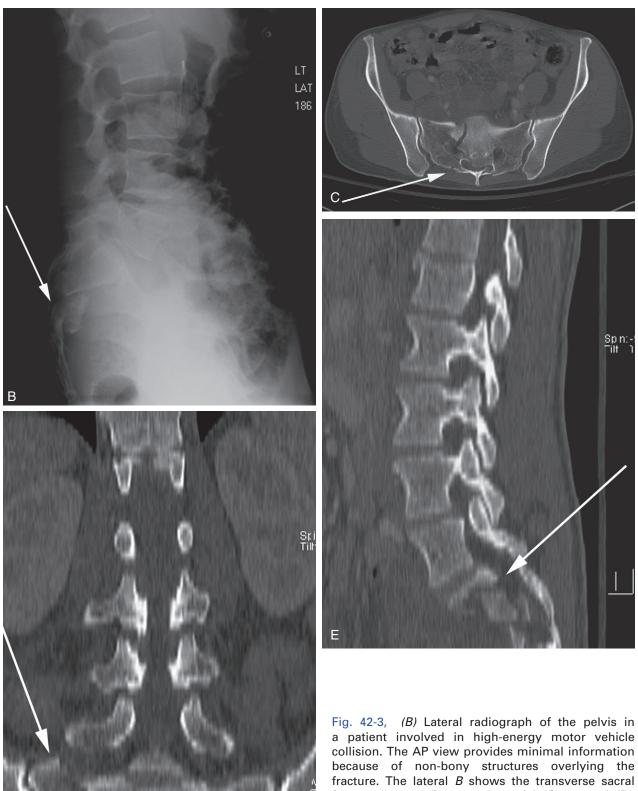
Fig. 42-3 *A*, Preoperative AP radiograph.

Fig. 42-3, *(B)* Lateral radiograph of the pelvis in a patient involved in high-energy motor vehicle collision. The AP view provides minimal information because of non-bony structures overlying the fracture. The lateral *B* shows the transverse sacral fracture *(arrow).* Preoperative axial *(C),* coronal *(D),* and sagittal *(E)* views of the sacrum on CT. The *arrow* denotes the fractures region. CT, with multiaxial re-construction, is the modality of choice for evaluation of sacral fractures.
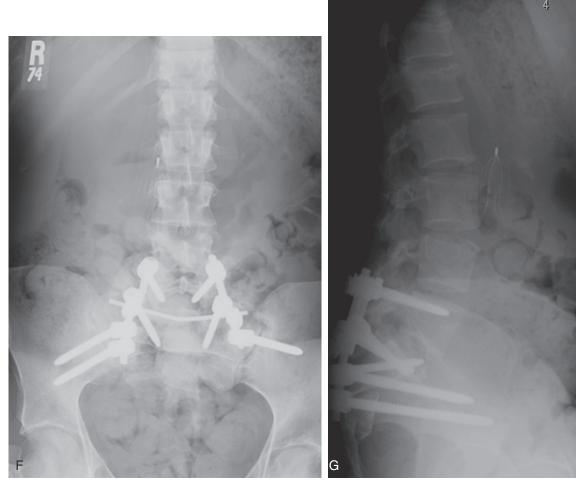
Fig. 42-3, Postoperative AP *(F)* and lateral *(G)* views of the pelvis and sacrum. Fixation of the sacral fracture with iliac and lumbar screws with interconnecting rods provides for a biomechanical stability and facilitates early mobilization.

conjunction with other physicians participating in the patient's care to properly prioritize needed therapeutics.

General indications for operative treatment include presence of neural compression with neurologic deficit, extensive posterior lumbosacral disruption, and fractures causing soft tissue compromise.[3] Relative indications for emergent operative management include polytrauma in which early stabilization will facilitate mobilization and rehabilitation. Goals of operative therapy include fracture reduction and stabilization, realignment of the lumbo-sacropelvic alignment, stable and adequate soft tissue coverage, and neurologic decompression if needed.[3] Proper stabilization can, in turn, improve mobilization, protect neurovascular structures, and decrease pelvic pain.[15,16] Early operative fixation has been associated with more accurate reductions and with better neurologic outcomes (in the presence of neurologic impairment at the time of injury).[2,16] Delayed diagnosis and inadequate treatment can lead to painful deformity and continued neurologic decline.[17]

Relative contraindications to definitive operative fixation include significant soft tissue compromise in the surgical field and other organ system or systemic dysfunction.

Operative intervention in a patient that has not been adequately resuscitated or medically stabilized increases the risk for infection, blood loss, and break down of soft tissue coverage.[5]

There are no absolute indications for neurologic decompression because there is little evidence that the natural history of the neurologic lesion is clearly altered with intervention.[18] However, if a decompression is chosen it should be performed early (within 24 to 72 hours from time of injury) because increasing epineural fibrosis and scarring of the foramina and central canal with time will increase the risk involved with the procedure.[18,19] Decompression can be achieved indirectly by fracture reduction or directly by focal foraminotomy or laminectomy.

In addition to neurologic injuries, one must also consider two other intimate structures (the pelvis and lumbar spine) that are commonly traumatized along with the sacrum and, therefore, factor into the decision-making process. If a sacral fracture presents as part of an unstable pelvic injury, initial reduction and stabilization of the pelvic ring should be achieved with anterior external fixation,

a posterior pelvic clamp, pelvic binder, or skeletal traction. Definitive management is typically operative and should be based on the goal of maintaining proper pelvic ring continuity and stability. If the sacral fracture has no associated neurologic deficit, pelvic instability, or significant soft tissue compromise, then nonoperative management is an option. However, there are very few data comparing the outcomes of operative and nonoperative therapies in this population.[3,20]

Transforaminal sacral fractures (especially if displaced) should raise the suspicion for a concomitant lumbosacral injury. Surgical stabilization is indicated if there is evidence of lumbosacral instability or facet dislocation on CT. Although evidence-based data comparing treatment options are lacking, unstable facet injuries are known to lead to symptomatic residual incongruence.[21,22] More significant displacing energy can lead to lumbosacral dislocation or traumatic spondylolisthesis, a rare and often fatal injury.[23] Anterolisthesis of L5 on S1 can occur through bilateral dislocation of L5-S1 facet joints, fracture-dislocations of those joints, or a fracture through S1. Surgical treatment for these rare disruptions, although necessary, is not well defined.[23] High-energy impact can also cause spondylopelvic dissociation, a rare U-shaped injury that results in disruption between the lumbar spine, sacrum, and remainder of the pelvis. Surgical fixation is indicated and includes percutaneous screw fixation, a modified Galveston technique, or lumbar spine to pelvic fixation.[13,24–26]

FRACTURES OF THE COCCYX

Because structural integrity of the coccyx does not contribute to pelvic stability (Tile A3 type fracture), there is little role for operative fixation of these injuries.[20] Significant anterior displacement of the coccyx could signify tenting of, or penetration into, the anal region. In such cases, closed digital reduction or operative treatment (in the case of an open fracture) may be indicated.[10] The mainstay of treatment is symptomatic in nature and includes limiting direct pressure to the area, pain control, and coccyx-unloading cushions. If pain and discomfort are severe or persist, coccygectomy is indicated for relief with good results.[27]

OPERATIVE PROCEDURES AND TECHNIQUE

SELECTION OF FIXATION METHOD AND IMPLANT

Methods for operative sacral fracture stabilization include anterior pelvic fixation, iliosacral screws (least invasive), direct posterior plating, and lumbo-pelvic fixation (most stable). Prior to fixation of the sacral fracture, proper reduction and stabilization of the anterior pelvis will facilitate the sacral procedure by providing some increased stability to the pelvic ring. The ideal sacral fixation implant will provide adequate stability and rigidity while creating minimal soft tissue disruption during implantation of the fixation. Percutaneous techniques are therefore preferred, as long as they impart adequate stability.

ILIOSACRAL SCREWS

Iliosacral screws provide appropriate fixation for primarily vertical disruption patterns that do not exhibit significant comminution, aberrant anatomy, or displacement not amenable to closed reduction.[28] These screws can be placed percutaneously under fluoroscopic guidance with the patient positioned either prone or supine; this flexibility is advantageous when treating the multiply injured patient. Intraoperative neuromonitoring studies have shown the potential for iatrogenic nerve damage, especially with incomplete reduction.[29–31] Other dangers include injury to gastrointestinal structures, although the most common complications are malreduction or loss of reduction. With proper technique and intraoperative imaging, however, the safety of this technique has been well established.[4,12,13] There is also growing interest in use of computer-assisted fluoroscopic navigational assistance in placing percutaneous iliosacral screws. Although data are preliminary, several studies have shown increased accuracy and decreased fluoroscopy exposure time with the use of navigation systems.[32,33]

The screw is placed across the sacroiliac joint from a lateral to medial direction under fluoroscopic guidance through stab skin incisions. The tip of the screw should be aimed toward the "safe zone" between the S1 foramen and the superior margin of the ala (visualized on outlet view) and the neural canal and anterior margin of the body (visualized on inlet view) (Fig. 42-4). Overtightening of the screw should be avoided.

POSTERIOR PLATING

Compared with iliosacral screws, posterior plating offers better fracture visualization, the opportunity for direct fracture reduction, and better stability for a variety of fracture patterns. Disadvantages include the necessity for a prone operative position and postoperative soft tissue complications. For this technique, the patient is placed in a prone position. For primarily lateral fracture patterns (or if a concomitant sacroiliac injury is present), a vertical incision is made 1 cm lateral to the posterior superior iliac spine from the level of the crest proximally to the level of the sciatic notch distally (Fig. 42-5, *A*). Following subcutaneous dissection, the gluteus maximus is released along its superior and medial margins and reflected anterolaterally, thus exposing the lateral sacrum (Fig. 42-5, *B*). Direct visualization and reduction of the fracture can be achieved and neural decompression can be performed as necessary. The addition of a contralateral approach allows
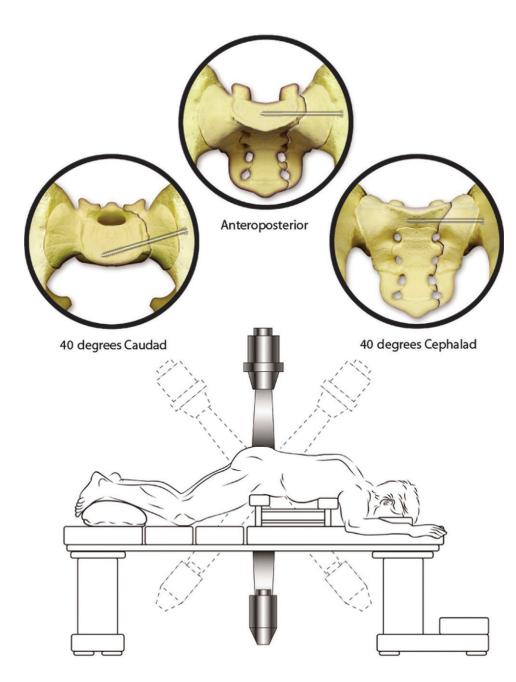
Fig. 42-4 For this technique, the patient may be prone or supine. The screw is placed across the sacroiliac joint under fluoroscopic guidance through stab skin incisions. The tip of the screw should be placed between the S1 foramen and the superior margin of the ala (40 degree cephalad view) and the neural canal and anterior margin of the body (40 degree caudad view).(Adapted from Matta JM, Saucedo T: Internal fixation of pelvic ring fractures. Clin Orthop 242:83, 1989.)

for a construct that spans both sacroiliac joints. For transverse or more central fractures (e.g., zone 3), a midline incision that runs from the L4 spinous process to the level of S4 is used. After adequate exposure is obtained by subperiosteal dissection and detachment of paraspinal muscles, direct visualization and reduction of the fracture is achieved. Laminectomy centered about the fracture line can aid in reduction and provide neural decompression if needed (Fig. 42-6). After fracture reduction is achieved, precontoured reconstruction plates are applied in the orientation best suited for the fracture pattern (see Fig. 42-6). The direct midline approach can be associated with a greater risk of soft tissue complications as this area may be involved during the initial trauma.

OTHER TECHNIQUES

The anterior approach with subsequent fixation (Fig. 42-7) is the least desirable method to treat sacral fractures; it is the most invasive of the approaches, places vital abdominal/pelvic contents at risk, and provides little flexibility in options for decompression or fixation. The most stable biomechanical construct provides rigid control of the lumbar spine, sacrum, and ilium and maintains stability, even with complete nerve root decompression. This construct uses pedicle screws in the lower lumbar spine and S1 connected by longitudinal rods along with fixation to the ilium bilaterally via the Galveston technique (Figs. 42-3, F and G).[34] In addition, a posterior L5-S1 fusion can be used to treat unstable lumbosacral junction injuries.[35,36]
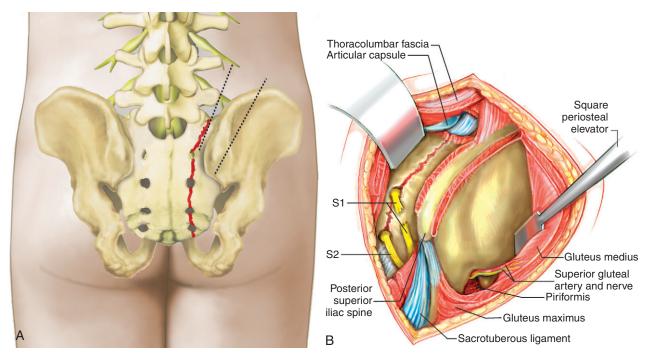
Fig. 42-5 *A,* An incision is made 1 cm lateral to the posterior superior iliac spine from the level of the crest to the level of the sciatic notch. *B,* Following subcutaneous dissection, the gluteus maximus is released along its superior and medial margins and reflected anterolaterally exposing the lateral sacrum. Direct visualization and reduction of the fracture can be achieved and neural decompression can be performed as necessary. (Adapted from Tile M, Helfet DL, Kellam JF: Fractures of the Pelvis and Acetabulum, 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2003.)
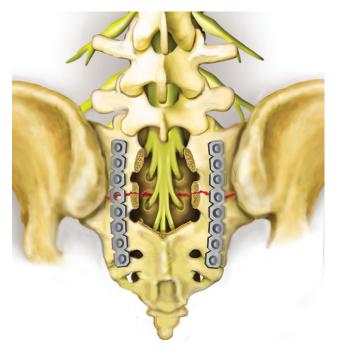
Fig. 42-6 Exposure is obtained though a midline incision that runs from the L4 spinous process to the level of S4, subperiosteal dissection and detachment of paraspinal muscles. Laminectomy should be centered about the fracture line. After fracture reduction is achieved, precontoured reconstruction plates are applied in the orientation best suited for the fracture pattern. (Adapted from Browner BD, Jupiter JB, Levine AM, et al: Skeletal Trauma: Basic Science, Management, and Reconstruction, 3rd ed. Philadelphia, WB Saunders, 2003.)

APPROACH TO THE COCCYX

To access the coccyx, the patient is placed in a hip-flexed prone position on the operating table. A straight midline incision from the distal sacrum over the coccyx and into the proximal portion of the crease of buttocks is used. Midline approach is continued to the level of the fracture with subsequent periosteal dissection around the coccyx. If significant anterior displacement is encountered, care must be
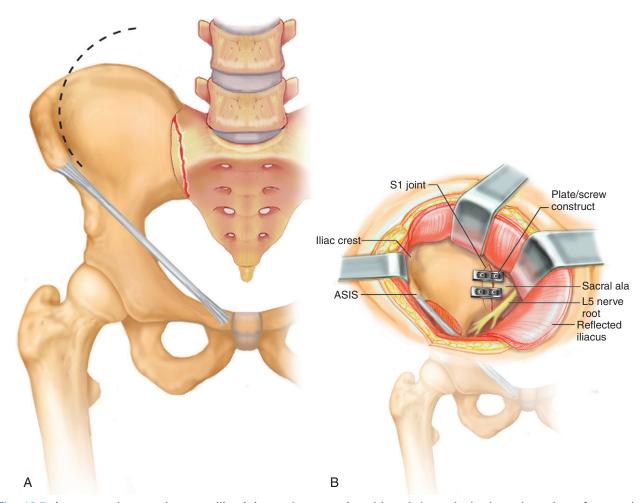
Fig. 42-7 Access to the anterior sacroiliac joint and sacrum is achieved through the lateral portion of a standard ilioinguinal approach to the pelvis. Dissection through elevation is carried out until the fracture site is adequately exposed for plate application. (Adapted from Schatzker J, Tile M: Rationale of Operative Fracture Care, 2nd ed. Berlin, Springer, 1996.)

taken to avoid injuring the rectum, the structure immediately deep to the coccyx.

OUTCOMES

Prospective data concerning sacral fractures following surgical fixation have not been reported. Retrospective studies reporting long-term outcomes are limited and short-term reports are heterogeneous in patient selection, surgical method, timing of surgery, reporting of neurologic injuries and post-surgical outcomes (making comparisons across studies difficult).[2,37] Published infection rates have a wide range from 5% to 50%. Neurologic deterioration is uncommon (range 2%–5%), fracture union occurs in the vast majority of cases (range 85%–90%), and pain can persist for approximately 30% of patients.[18,38–42] No significant series of surgically treated coccygeal fractures have been reported.

SUMMARY

Sacral fractures are usually the result of high-energy mechanisms, concomitant with other significant injuries, and delayed in diagnosis. Inadequate or delayed treatment can lead to continued pain and neurologic decline. Clinical suspicion warrants radiologic studies beyond plain radiography including a CT scan for detailed bony evaluation and an MRI for neural evaluation (if a deficit is present). When neurologic injury is present (highest in Denis zone 3 fractures), early operative decompression and fixation may improve neurologic outcomes and facilitate ease of surgery. It is important to optimize the patient's general medical status prior to surgery and to consider adjacent pelvic and lumbar injuries in the operative plan. Fixation methods range from percutaneous iliosacral screws to posterior lumbopelvic instrumentation and should be selected based on degree of stability required from the implant and

fracture pattern. In contrast, coccyx fractures are typically the result of low-energy trauma, do not present with neurologic deficits, and are amenable to symptomatic nonoperative management.

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Spinopelvic Fixation Techniques

INTRODUCTION

Fewer than 2% of all spine injuries affect the lumbosacral junction. Lumbosacral injuries include burst fractures, traumatic lumbosacral spondylolisthesis, and spondylopelvic dissociation.[1] Treatment of these injuries varies between the adult and pediatric population. In children, the majority of these injuries can be treated with closed reduction and immobilization in cast or brace, whereas in adults, these injuries might require spinopelvic fixation.[2]

Lumbosacral injuries are a result of extreme force and dissipation of kinetic energy and usually are caused by a combination of hyperflexion and rotation. Sacral promontory facet fractures and neurologic deficits commonly occur in conjunction with these injuries.[3] Spinopelvic trauma often is associated with thoracic, abdominal, pelvic, and cranial injuries. Identifying and managing emergent life-threatening issues is paramount. Initial high-quality anteroposterior and lateral view radiographs of the lumbosacral junction can delineate the spinal injury. If the initial radiographs are not closely scrutinized, these injuries can be missed. Advanced imaging modalities such as computed tomography and magnetic resonance imaging usually are required to identify and characterize lumbosacral injuries after the patient is stabilized.[4–6]

Fixation of the lumbar spine to the pelvis can be challenging. The lumbosacral transition is the least understood osseous structure in the spine. The lumbopelvic junction in humans has certain specific characteristics: the presence of structural lordosis and a range of movement in flexion and extension. In synergy with the hip joints, this articulation allows humans to maintain the trunk in a vertical position yet provide the flexibility to voluntarily assume a horizontal posture.[7,8]

RELEVANT ANATOMY FOR LUMBOSACRAL INTERNAL FIXATION

The lumbopelvic junction consists of the last three lumbar vertebrae, upper three segments of the sacrum, and sacroiliac joint. The lumbar segment is lordotic, mobile, and situated deep in the body. The sacral segment is rigid, convex in shape in the sagittal plane, and more superficial. Flexion and extension are 53 degrees and 30 degrees, respectively, providing a total arc of motion of 83 degrees at the lumbosacral junction.[2,7–9]

For placement of pedicle screws in the lumbar spine, the starting point is caudal to the superior articular facet, lateral to a vertical line drawn through the facet's base, and in line with the horizontal axis of the transverse process. This point becomes progressively more lateral as one proceeds distal in the lumbar spine. The lateral to medial angle of inclination of the pedicles increases from 7 to 14 degrees at L1-L2 and to 25 to 35 degrees at L5.[10]

Screw fixation in the sacrum can be challenging. S1 pedicle fixation can be achieved in several areas: medially into the S1 pedicle and body, laterally into the sacral ala, or straight into the ala at the S1 level. Asher and Strippgen[11] and Burton et al.[12] found the greatest volume of bone available in the sacral ala laterally in the region of the first sacral level. Zindrick and Lorenz[13] compared the three screw techniques and found the lateral ala to be slightly superior to the medial sacral site in pullout tests, although the difference was not statistically significant. Carlson et al.[14] found that anteromedially oriented screws combined with rigidly constrained instrumentation resulted in the greatest axial load to failure and the least degree of screw rotation. In a related study, Smith et al.[15] found that medially oriented sacral screws produced the highest pullout strength. They also found the bone density in the center of the sacral body to be superior to that of the sacral ala and to provide higher pullout strength. Luk et al.[16] recently showed that screws attaining bicortical purchase through the S1 endplate had significantly higher strengths than those placed through the anterior sacral cortex. They also found that insertion torque was a good indicator of pullout strength after cyclic loading. The most

common complication is injury to the traversing L5 nerve root if the screws are prominent anteriorly.[16]

The ilia are paired bones that connect the pelvis to the spine via the sacroiliac joints. The ilium also articulates with the ischial and pubic bones. Miller et al.[7] found the bony architecture of the ilium to be much like that of a long bone. The metaphyseal components of the ilium are located anteriorly and posteriorly, with the diaphysial section of the bone located in between. Iliac fixation typically has provided additional fixation points in long fusion constructs spanning the lumbosacral spine. Transverse rods or bars spanning the iliac crests have been used in combination with Harrington and Luque instrumentation for several decades.[7] Allen and Ferguson[17] developed the concept of placing rods into the body of the ilium. Recent techniques for iliac fixation use intrailiac screw fixation instead of rods.

Miller et al.[7] concluded that the ideal location for iliac fixation was in the center of the ilium, on a line that begins from a posterior entrance site on the medial aspect of the posterior ilium at the level of S2-S3 on the sacrum and passes 1 cm superior to the sciatic notch as it crosses the body of the sacrum. Asher and Strippgen[11] described the intrailiac entry site at the level of the S2 lamina in the transverse plane on the posterior superior iliac spine. After the intrailiac cortical bone is exposed, the screw is passed through the ilium toward the anterior inferior iliac spine. Olsen et al.[18] showed the biomechanical superiority of lumbosacral fixation when using the ilium. In tests of 10 different lumbosacral fixation systems, the devices that used either an iliac rod or an iliac screw had maximum stiffness and maximum moment at failure.

STRUCTURES AT RISK IN SPONDYLOPELVIC FIXATION

Numerous structures are at risk during sacral pedicle screw insertion at the lumbosacral junction. These structures include the dural sac, nerve roots, and great vessels anterior to the vertebral bodies and sacral ala. Penetration into the spinal canal at the time of pedicle screw insertion can cause injury to neural structures. A fractured pedicle or a medial breach can impinge the exiting nerve root or ganglion. Lateral screw placement can result in injury to the nerve originating from the level above as it runs lateral to the pedicle and anterior to the transverse process.[16,19,20] The difficulty with placing pedicle screws rises exponentially if the anatomy is significantly altered by previous surgery or pathologic processes. A thorough review of the preoperative computed tomographic scans will reveal rotational abnormalities, pedicle deformities, and the extent of altered posterior landmarks.[6]

The great vessels lie anterior to the vertebral bodies in spinal levels above their bifurcation. At the L4 vertebral body level, the aortic artery and inferior vena cava leave the midline, take a lateral position, and lie directly anterior to the

pedicles. In the sacral region, the great vessels (the internal and external iliac arteries and veins) lie laterally along the sacral ala.[21]

Iliac screw fixation can injure several vital structures in the pelvis. Medial penetration of the ilium can injure the iliopsoas muscle, abdominal viscera, and great vessels, leading to catastrophic complications. Lateral breach of the ilium damages the gluteal musculature. Screws advanced too deeply into the body of the ilium anteriorly risk acetabular injury. More posteriorly angulated iliac screws can violate the greater sciatic notch, injuring the sciatic nerve or the superior gluteal vessels.[21–23] With the appropriate understanding of anatomy and preoperative planning, instrumentation of the lumbosacral spine and pelvis can be a safe, effective tool with which to achieve rigid segmental fixation in this region.

LOW LUMBAR BURST FRACTURES

The treatment of unstable fractures and fracture dislocations of the lumbar spine remains a controversial issue. The concept of a burst fracture was first described by Holdsworth[24] in 1970 and was thought to represent a stable injury with intact anterior and posterior ligamentous complexes. In 1983, Denis[25] described such injuries to the thoracolumbar junction as being unstable and introduced the three-column theory of spinal stability. Fractures were classified as compression, burst, Chance, or fracture-dislocations. Later, the AO classification defined fractures patterns as compression, distraction, and fracture-dislocation. Numerous subtypes delineate increasing severity of injury, from compression to fracture-dislocation. Although the Denis and AO classifications provided useful descriptive terminology, they failed to address the best clinical treatment for a particular fracture pattern.[26] McCormack et al.[27] devised a load sharing classification that uses three separate characteristics of fractured vertebral body: amount of vertebral body comminution, apposition of fracture fragments at the fracture site, and amount of kyphotic deformity corrected. Based on the characteristics of the fracture, short- or long-segment posterior fusion is recommended. Although several classification systems have been described, a comprehensive classification system that has clinical application remains elusive.

Fractures of the low lumbar spine constitute fewer than 4% of all spine fractures. These uncommon injuries with unique biomechanical and anatomic characteristics should not be grouped with the more common injuries that occur at the thoracolumbar junction.[27–30]

Earlier descriptions of treatment of L5 burst fractures describe success with bedrest and subsequent bracing. In 1988, Levine and Edwards[31] presented a report of their short-term operative experience with L4 and L5 burst fractures treated with segmental posterior spine fixation. They noted excellent restoration of vertebral height, improved

lordosis, and successful fusion in all patients who were followed for more than 6 months.

Although certain burst fractures at the lumbosacral level can be treated nonoperatively, the primary indication for surgical intervention is injury affecting both the anterior and posterior elements with resulting instability and/or the presence of neurologic deficit. In addition, a sagittal plane deformity at the lumbosacral junction with kyphosis of 30 degrees or greater requires instrumentation and fusion to achieve and maintain lumbosacral lordosis.[11,12,21]

Low lumbar burst fractures can be treated from a posterior approach, which allows for decompression of any entrapped neural elements and a posterolateral arthrodesis with instrumentation. Even with compression anterior to the thecal sac, canal decompression can be accomplished by working around the cauda equina in the lower lumbar spine. For posterior instrumentation, pedicle screws and rods are used because of the rigidity of such constructs, and the number of levels required for stabilization is minimized when compared with the use of hooks. In most cases, fractures of the fifth lumbar vertebra can be stabilized by L4 to S1 fusion. For L4 fractures, it usually is sufficient to instrument and fuse from L3 to L5. Occasionally, with severe osseoligamentous disruption, extension of the instrumentation down to the sacrum and up to L2 might be needed. In addition, iliac screw fixation might be necessary in severely osteoporotic patients with L4 or L5 burst fractures.[23,32,33] An et al.[34] presented a report of 20 patients with low lumbar burst fractures, 7 of which were treated nonoperatively and 13 of which were treated operatively by using a posterior approach. The average follow-up durations were 56.2 months and 39.0 months for the conservatively and operatively treated groups, respectively. The authors found no evidence of significant loss of reduction or late neurologic compromise in the surgical group. They reported an average follow-up kyphosis of 9.2 degrees and 31% loss of vertebral height in the conservative group compared with an average follow-up kyphosis of 1 degree and 19% loss of vertebral height in the surgical group. They concluded that conservative management was a viable option for neurologically intact patients and recommended short-segment fixation with pedicular instrumentation in favor of long fusion with distraction instrumentation, because patients with longer fusions were found to have more disabling back pain. Benzel and Ball[35] proposed a posterior decompression and fusion technique that uses S2 dorsal neuroforamina hook fixation as an alternative to sacral screw or iliac fixation for the treatment of low lumbar fractures requiring fusion to the pelvis. They reported attaining a solid fusion in six of six cases. Seybold et al.[36] showed that posterior short-segment transpedicular instrumentation maintained vertebral height and prevented kyphotic collapse as well as anterior instrumentation and corpectomy in their series in which 22 patients with low lumbar burst fractures were treated operatively. They also showed that posterior procedures are generally fast and less technically demanding than anterior procedures, particularly in the low lumbar region.

The anterior approach should be reserved for situations in which severe comminution of the L5 vertebral body is present and posterior stabilization alone might be inadequate to provide stability or when neural compression requires anterior decompression. Anterior decompression or fusion is achieved via an anterior transperitoneal or retroperitoneal approach. Restoration of anterior column stability usually can be achieved by a strut graft/cage extending from L4 to the sacrum. Placing anterior instrumentation in this region is difficult because of the proximity of the major blood vessels and the difficulty in achieving secure fixation with an anterior plate construct spanning the lumbosacral junction.[22,37]

Huang et al.[38] reported their experience with the anterior locking plate, comparing seven patients who underwent anterior decompression and plating and seven patients who underwent posterior surgery with decompression, posterolateral fusion, and short-segment instrumentation for lower lumbar fractures. They noted that the average neurologic recovery was 1.1 Frankel grades for the anterior locking plate group versus 0.9 for the posterior surgery group. They also found a higher rate of hardware failure in the posterior surgery group compared with the anterior locking plate group.

Endoscopic techniques for gaining anterior access to the low lumbar region have been reported.[38] Olinger et al.[39] reported their experience with an endoscopic retroperitoneal approach for anterior decompression and fusion of lumbar spine fractures from T12 to L5. They reported no major complications and minimal blood loss. None of the 11 patients in their study required conversion to an open procedure, and all were able to be mobilized as early as the second postoperative day.

The problems associated with instrumenting the lumbosacral spine have been well documented. Surgery in this region has been associated with a high rate of pseudarthrosis. Grubb and Lipscomb[40] reported a pseudarthrosis rate of 35% for non-instrumented lumbosacral fusions versus 6% for instrumented fusions. Lonstein[41] indicated that arthrodesis at the lumbosacral junction is placed under high stress, considering the fixed nature of the spine and the innate mobility of the pelvis. Based on the high level of stress, a combination of anterior and posterior procedures provides the best environment in which to achieve a solid lumbosacral arthrodesis. Gertzbein[42] and Gertzbein et al.[43] reported a 100% fusion rate at an average follow-up duration of 2.7 years when using a circumferential fusion technique in 25 patients who underwent previous lumbar fusion surgery and went on to develop pseudarthrosis.

TRAUMATIC SPONDYLOLISTHESIS

Watson-Jones[44] was the first to describe lumbosacral dislocation, which he attributed to a hyperextension mechanism. Most authors consider a combination of hyperflexion and

compression to be the most common mechanism to produce bilateral L5-S1 dislocation. Roaf [45] showed experimentally that a combination of hyperflexion with vertical loading and rotation is necessary to create this injury. Hyperflexion alone is not capable of producing either pure dislocation or fracture-dislocation in the lumbar spine.

Herron and Williams[46] suggested that the presence of lumbar transverse process fractures should lead to the suspicion of traumatic lumbosacral dislocation. In addition, the existence of a perineal laceration mandates radiographic examination of the lumbosacral spine to exclude the occurrence of L5-S1 dislocation.

Several radiographic clues seen on anteroposterior and lateral view radiographs of the lumbar spine can lead to the diagnosis of lumbosacral injuries. The anteroposterior view can show widening of the paravertebral soft tissue lines, multiple transverse process fractures, widening of the interpedicular distance, and rotational deformity of the spinous processes. The lateral view should be scrutinized for the disruption of the spinolaminar lines, anterior or posterior subluxation of L5 on S1, exaggeration of lumbar lordosis, and widening of the interspinous process distance. Accurate differentiation of a preexisting isthmic spondylolisthesis from lumbosacral fracture dislocation is suggested by identifying a chronic defect in the pars interarticularis. Differentiation from degenerative spondylolisthesis is somewhat more difficult. All reported cases of acute lumbosacral fracture dislocations have shown at least 20% subluxation at the L5-S1 junction, whereas degenerative spondylolisthesis generally is associated with less displacement.[5,6,28,47]

Newell[20] reported success with brace treatment for a lumbosacral dislocation in the unreduced position. Recently, Veras del Monte and Bago[48] reported the long-term follow-up of a patient who was treated nonoperatively. The authors reported that at the 10-year follow-up, the patient had only mild pain and maintained normal lumbar spine mobility.

Most authors think that lumbosacral dislocation is a three-column injury and that it consequently is highly unstable. Closed reduction often is unsuccessful and often requires prolonged traction. Surgical treatment of these injuries has been primarily through the posterior approach. Vialle and Court[49] presented the reports of four cases of lumbosacral dislocation, all of which were treated with a posterior approach and transpedicular fixation. In one case, the posterior construct was supplemented with an anterior fusion through a retroperitoneal approach with an interbody cage. Two patients were fused from L5 to S1, whereas the other two patients underwent fusion from L4 to S1. At long-term follow-up, three of the four patients were asymptomatic. The one patient who required anterior fusion reported occasional pain at the lumbosacral joint. It should be noted that all these patients were neurologically intact throughout their course. Cruz-Conde et al.[50] presented the report of a 42-year-old man who had

been involved in a motor vehicle accident and who sustained an anterior L5-S1 dislocation. He was treated with open posterior reduction, internal fixation from L4 to S1, and fusion. The authors reported that complete fusion was achieved and that at the 5-year follow-up visit, the patient was asymptomatic.

Partial facetectomy can be performed in cases of dislocation to facilitate reduction. However, intact facet joints serve as a block against deforming forces that can cause redislocation.[51] Decompressive laminectomy generally is not indicated in the absence of neurologic signs and can create further instability.[50,52–55] The anterior approach to traumatic spondylolisthesis has been reserved for cases in which posterior approach is compromised. In two reported cases of anterior fixation, both patients had open posterior lumbosacral injuries. Contaminated wounds prohibited initial posterior approach and fixation. In both cases, the anterior fixation was later supplemented with posterior fusion and instrumentation.[1,14,22] In the report presented by Carlson et al.,[14] anterior fixation was attempted with an L5-S1 transfixing fibular dowel. The patient subsequently fractured the fibular dowel and experienced loss of reduction and neurologic deterioration. Later, posterior fusion with pedicle screw fixation, intrasacral rods, and iliac crest autograft was used for stabilization.

TRAUMATIC SPONDYLOPELVIC DISSOCIATION

Traumatic spondylopelvic dissociation is a distinct injury pattern characterized by a transverse sacral fracture in conjunction with bilateral sacroiliac fracture dislocation. Traumatic spondylopelvic dissociation results in mechanical dissociation of the pelvis from the spine as the result of a highly comminuted sacral fracture with severe instability in the cephalad direction.[56–58] Options for the surgical treatment of spondylopelvic dissociation are limited in that the sacrum might not provide structural support or stability for internal fixation. Vresilovic et al.[58] described a modified Galveston iliac fixation technique. Originally, the Galveston technique was accomplished by inserting angled rods into the iliac wings. Vresilovic et al.[58] described spanning fixation from the lumbar spine directly to each ilium with the use of screws and rods. Placing two iliac screws per ilium and connecting them both to the distal rods allows a greater degree of stability, and an improved ability to mobilize the patient was achieved.

Bents et al.[59] reported a case of severe trauma with complete displacement of the lumbar spine into the pelvis. The fracture was stabilized with a pedicle screw construct attached to the pelvis by using the Galveston technique. Bilateral iliac screws were used to increase stability. At the 2-year follow-up visit, the patient was pain-free, ambulating in an ankle-foot orthotic, and had persistent deficit involving the right L5 and S1 nerve roots.

PEDIATRIC LUMBOSACRAL INJURIES

Unstable traumatic lesions of the lumbopelvic junction in children are rare; only a few cases have been reported. For the most part, these injuries occur in teenagers and are treated in a manner similar to the treatment used for adults. With spinopelvic fixation for trauma, some concerns are unique to the pediatric population. Children, depending on their age and stage of development, have future growth potential. Additionally, absolute sizes of the anatomic structures generally are diminutive compared with those of adults. The soft tissue structures have greater elasticity in children compared with adults. The thicker periosteum of children is much more active in a biologic sense than that of adults. In addition to increased elasticity of soft tissue structures, the bony structures of children can absorb more energy before reaching ultimate failure. This is partly because cartilage makes a greater contribution to bone volume in children than in adults.[42,57,60,61]

In 1995 Beguiristain et al.[60] presented the report of a 5-year-old boy who sustained a traumatic bilateral L5-S1 dislocation with associated L5 and S1 nerve root deficits. He was treated with traction, closed reduction, and postreduction plaster immobilization. At the 8-year follow-up visit, no neurologic deficit was evident and adequate lumbosacral alignment was present. It should be noted that no fracture was associated with the dislocation. Novkov et al.[61] presented the report of a 12-year-old boy who sustained an S1 fracture-dislocation that was treated with closed reduction and traction. Although the authors reported some loss of initial reduction and 13 months of bladder and bowel dysfunction, the neurologic deficits eventually resolved with no functional sequelae from the loss of reduction.

Most adolescent spinopelvic injuries are treated similarly to their adult counterparts. In 1992 Connolly et al.[51] presented the reports of two adolescent patients with fracture-dislocation of the lumbosacral junction. The results of the neurologic examinations were normal for one child but revealed sensory deficit for the other. Both cases were managed by open reduction and internal fixation via a posterior approach with good long-term follow-up. Similarly, Gertzbein[42] presented the report of a 19-year-old patient who presented with a posterior dislocation of L5-S1. Unilateral motor deficit was noted. Open reduction with posterior pedicle screw instrumentation was performed.

Hanley et al.[57] presented the report of an adolescent patient with a complex lumbopelvic fracture-dislocation. Complete anterior and inferior fracture dislocation of S1 and S2 was present along with a longitudinal sacral fracture through the neuroforamen. Partial motor deficit was present. Reconstruction was performed by first stabilizing the longitudinal sacral fracture with posterior transiliac fixation. Lumbar pedicle screw instrumentation was then used to achieve a distractive reduction with restoration of sacral alignment.

Bilateral facet dislocation of the lumbosacral junction is a rare injury. Finkelstein et al.[62] presented a report of a 16-year-old male pedestrian who was struck by an oncoming train who sustained an open fracture dislocation of the lumbosacral junction with L5 vertebrae completely behind the sacrum. Because of posterior contamination, an anterior approach was used with plating and then delayed posterior fusion with instrumentation.

CONCLUSION

Injuries to the lumbosacral region are infrequent and result from high-energy trauma. The treatment of these injuries differs between children and adults. Most low lumbar burst fractures, lumbosacral spondylolisthesis, and spondylopelvic dissociations require spinopelvic fixation in adults, usually with a posterior approach. In the pediatric population, these injuries can be successfully treated with traction, fracture reduction, and immobilization.

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C H A P T E R

44

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Development and Maturation of the Axial Skeleton: Developmental Abnormalities of the Cervical Spine

INTRODUCTION

Developmental abnormalities of the cervical spine vary widely. Many of these anomalies are asymptomatic and go undetected, but several types may result in biomechanical instability and thus place a patient at risk for either neurologic injury or chronic pain from deformity. Identifying the lesions with significant clinical implications is important not only for treatment of the malformation itself, but because they may be associated with other nonspinal faults of development. Here we discuss the embryologic origins of the cervical spine, the congenital abnormalities associated with aberrations in development, and their clinical relevance.

GENETICS AND EMBRYOGENESIS

The development of the axial skeleton is complex, requiring the coordinated expression of a family of *homeobox* or *Hox* genes.[1] This family of genes has been highly conserved across several species with the majority of research being performed on *Drosophila melanogaster*. Normal expression of these genes results in the proper cranio-caudal orientation, segmentation, and growth of the spine.[2–4] Aberrant expression of these genes may be responsible for developmental abnormalities of the spine, although there is some redundancy in mammals so that a single genetic defect may not result in a significant deformity.[5] The disruption of selected *Hox* genes may result

not only in defects in the patterning of the axial skeleton but also in flaws in cell development.[6–9] For example, defects in *Hox* gene expression have been associated with neuroblastoma, Wilms' tumor, and leukemia—tumors that have a known association with vertebral column anomalies.[6,7,9] In mice, several authors have described an association between the development of cervical ribs and particular malignancies including leukemia and fibrosarcoma.[7,10–12] An association between cervical rib formation and childhood malignancy has also been suggested in the literature.[2,13]

The embryologic chronology of axial spine development is divided into six overlapping stages. The first stage, gastrulation, occurs 2 weeks after fertilization of the embryo. In this stage, the embryo assumes three layers; endoderm, mesoderm, and ectoderm. During early gastrulation, the primitive streak forms at the caudal end of the embryo. Ultimately, the primitive streak begins to regress toward the caudal pole. Cells from the deep surface of the primitive streak give rise to mesenchyme, which forms mesoderm. This stage, also referred to as the mesenchymal stage, is characterized by notochord development in the third week of embryonic development from cells within Hensen's node—the cranial extension of the primitive streak. The mesoderm condenses around the notochord and neural tube (paraxial bar) and develops lateral, intermediate, and paraxial proliferations. The lateral plate gives to pleural pericardium, peritoneum, mesentery, blood vessels, and limb-bud mesoderm. The intermediate mesoderm gives rise to the urogenital system. The paraxial mesoderm becomes the vertebrae, ribs, dermis, and musculature. In this second stage, mesenchyme differentiates out of the mesoderm into segmented somites.[14] These somitic cells migrate just caudal to Hensen's node and form 42 to 44 paired structures on either side of the notochord. The patterning of somites is determined by the expression of *Hox* genes. In the fourth week of embryonic life, the third stage of development commences as the somites differentiate into the sclerotome and dermomyotomes (Fig. 44-1). The bilaterally paired sclerotomes will give rise to a single vertebral body, migrating dorsally to form the posterior elements including pedicles and lamina, and ventrally to form the vertebral body and disks. The formation of the ventral parts of the vertebrae is under control of the *Pax* family of genes,
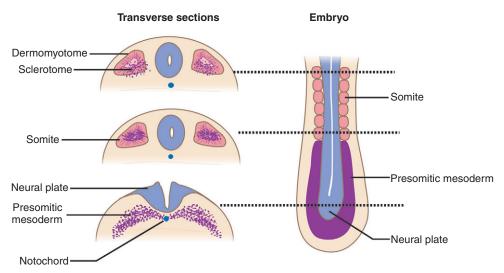
Fig. 44-1 Somitogenesis is depicted with unsegmented presomitic mesoderm giving rise to the paired somites. Each somite further divides into the sclerotome and the dermomyotome. The sclerotome will give rise to the vertebral body, whereas the dermomyotome will give rise to the subcutaneous tissue, dermis, and muscles of the dorsal trunk.

specifically *Pax*-1 and *Pax*-9. During chondrification (see below), this gene is also expressed in the developing disks. The dermomyotome, under control of *Pax*-3 and *Pax*-7, will form the dermis and subcutaneous tissues of the back and the muscles of the dorsal trunk.[15]

At 6 weeks, the fourth stage begins as the sclerotomes undergo a process of resegmentation that results in the chondrification of the notochord and formation of intervertebral disks. Here, the caudal half of one sclerotome fuses with the cranial half of the adjacent sclerotome (Fig. 44-2). This occurs while the mesoderm of the sclerotome is migrating ventrally to surround the notochord. The combination of these events results in the formation of a vertebral body with intervertebral disks. Each sclerotome develops a central cleft known as the fissure of von Ebner. The sclerotomal cells cranial to the fissure contain loosely packed cells, while cells caudal to the fissure are denser. This craniocaudal organization of the sclerotome is related to axonal formation as outgrowth of the spinal nerves is restricted to cranial portion of the sclerotome.[16,17] Cells from the fissure of von Ebner migrate to and around the notochord and are precursors of the intervertebral disk. The fifth stage is characterized by the chondrification of mesenchymal vertebral anlagen which occurs in dual centers on either side of the notochord and in the base of the lamina.

Chondrification also starts in the sixth embryonic week and is under control of the notochord and ventral neural tube. There are three paired centers for chondrification in each vertebral body. The first pair surrounds the notochord ventral to the neural tube and forms the center of the vertebral body. The second pair forms dorsolaterally and migrates dorsal to the neural tube to form the posterior vertebral arches and spinous processes. The third pair develops
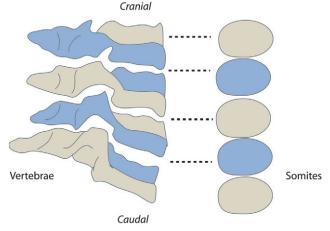
Fig. 44-2 Resegmentation is depicted with cells from the caudal half of one sclerotome fusing with the cranial half of the adjacent sclerotome to form the vertebral body.

between the ventral and dorsal pairs and forms the transverse processes. Chondrification starts at the cervicothoracic junction and extends cranially and caudally. Cells from around the notochord will condense around the notochord to produce the annulus fibrosus of the intervertebral disk. Physaliphorous cells of the notochord will form the nucleus pulposus.

The sixth stage is ossification of the vertebral column. Ossification of the spinal column in utero occurs in five centers (the centrum of the vertebral body, the two neural arches, and the two costal processes) and replaces the chondrified vertebral anlagen. Ossification begins in the 8th embryonic week, and ossification centers are visible in the embryo by the

14th week. Three ossification centers are generally agreed on by most authors, one in the center of the vertebral body, and two on either side in the dorsal vertebral arches.[18] Between three and six secondary ossification centers have been suggested in the dorsal arches to form the pedicles, laminae, and spinous processes. Ossification of the vertebral body begins at the thoracolumbar junction and spreads cranially and caudally. Ossification of the dorsal elements begins simultaneously from C1-L1 and proceeds caudally. The process of ossification does not end until some time between the 14th and 18th year of life.

The development of the craniovertebral junction is distinct from the subaxial spine. The craniovertebral junction develops from the fourth occipital sclerotomes (forming the occipital bone, clivus and occipital condyles) and the first and second cervical sclerotomes (forming the condyles and the ring of the foramen magnum) (Fig. 44-3). The anterior arch of the atlas is derived from the fourth occipital sclerotome and the posterior arch is derived from the fourth occipital sclerotome and the first cervical somite. The apical, transverse cruciate, and alar ligaments are also derived from the fourth occipital sclerotome. The axis is derived from the fourth occipital and the first and second cervical sclerotomes. The ventral portion of the first cervical sclerotome forms the majority of the base of the odontoid process. The rostral tip forms from the fourth occipital sclerotome. The body of the axis is formed from the second cervical sclerotome. Ossification of the axis occurs from six ossification centers. The odontoid process has paired ossification centers that fuse at 3 months postnatally. The tip of the odontoid process has a single ossification center. The body of C2 has three ossification centers, similar to the subaxial spine, which form the vertebral body and the dorsal elements independently. The

odontoid process fuses to the body of the axis beginning at 4 years of age and continues through age 8. The rostral tip of the dens fuses to the odontoid process by 12 years of age.

Despite an ever-increasing understanding of both the genetic and embryologic complexities of the development of the spine, specific genetic mutations or disruptions of specific embryologic events rarely correlate with a known congenital spinal anomaly. Reduced expression of *Pax*-1 may result in fusions between adjacent levels, but this phenomenon is also seen in normally occurring fusions such as fusion of the dens with the body of C2.[19] Environmental factors, such as fetal alcohol syndrome, maternal hypotension, and vascular insufficiency, have been implicated in some congenital malformations, such as Klippel-Feil syndrome (KFS), but have not had much further support.[4,20,21]

EPIDEMIOLOGY OF CONGENITAL DISORDERS

Because of the asymptomatic nature of many congenital abnormalities of the cervical spine, the true incidence is likely largely underreported. It is estimated that up to 5% of fetuses have vertebral anomalies.[22] Indeed, the true incidence of a relatively common congenital disorder of the cervical spine, Klipped-Feil Syndrome (KFS), is unknown because of the asymptomatic nature of the disease and a lack of other consistent anomalies.[23] Someauthors have reported that congenital anomalies of the cervical spine occur in approximately 1 in 40,000 to 1 in 42,000 births with a slight female predominance.[24,25]

The prevalence of congenital fusions of subaxial cervical vertebra based on the study of skeletal specimens has been reported as 0.71%.[26] Congenital fusions can occur at any level of the cervical spine with 75% occurring in the region
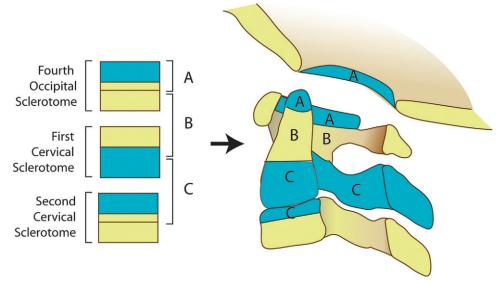

Fig. 44-3 Formation of the craniocervical junction and the upper cervical spine is depicted with fusion of the fourth occipital sclerotome and first and second cervical sclerotomes.

of the first three cervical vertebrae. Lower cervical fusions may occur in association with certain syndromes. Fetal alcohol syndrome has been associated with congenital neck fusions in up to 50% of patients affected by the disorder.[25]

Although many developmental abnormalities of the cervical spine are asymptomatic and incidental, those that may require treatment, such as associated instability or spinal canal stenosis, must be recognized for appropriate care.

DIAGNOSTIC EVALUATION

The cornerstone of diagnosing a cervical spine anomaly is plain radiography. This should include a lateral, anteroposterior, and open-mouth odontoid view. Plain radiographs may be difficult to interpret in children, however, given the variable patterns of ossification. If instability is suspected, flexion and extension views should be obtained. Computed tomography (CT) scans are useful in the diagnosis of cervical spine anomalies and can be performed even in the youngest of children. The advantages over plain radiographs include the ability to format sagittal and coronal reconstructions. Axial images may show an infringement of a bony structure into the spinal canal. Magnetic resonance imaging (MRI) is the study of choice for assessing soft tissues, including ligaments, and the spinal cord itself. If neurologic symptoms are reported or are suspected, an MRI should be performed to evaluate the integrity of the spinal cord.

UPPER CERVICAL SPINE ABNORMALITIES

The upper cervical spine is anatomically and biomechanically distinct from the remainder of the subaxial spine. Unlike the subaxial spine in which axial weight is transferred primarily through the vertebral body, the weight of the skull is transferred via the occipital condyles to the lateral masses of C1 and C2. There are no intervertebral disks at these levels. The primary motion at the occiput-C1 joint is flexion/extension, whereas the primary motion at C1-C2 is rotation. Several developmental abnormalities may occur at the craniovertebral junction.

OCCIPITALIZATION OF THE ATLAS

This condition is characterized by fusion of the occiput to the atlas and occurs in about 0.25% of the population.[27] It is generally defined as a failure of segmentation between the last (fourth) occipital and first cervical sclerotomes. It has been associated with various syndromes including achondroplasia, spondyloepiphyseal dysplasia, Larsen's syndrome, and Morquio's syndrome. Although it may be isolated, it usually occurs in conjunction with other anomalies such as congenital fusion of the second and third cervical vertebrae, basilar invagination, Chiari I malformation and Klipped-Feil Syndrome (KFS).[28,29] The original description of occipitalization of the

atlas indicated that the majority of cases were fused anteriorly with hypoplastic or anomalous posterior elements.[27,30] Many of these patients were symptomatic likely from instability resulting from a weakened or absent transverse atlantal ligament.[31] Symptoms described in these patients include weakness, numbness, or pain in the upper extremities with associated upper motor neuron signs including hyperreflexia and spasticity. Clinically, these patients may present with a low hairline, restricted neck movements, and short neck, all of which are also associated with Klipped-Feil Syndrome (KFS).[4,28] (see later).

A rarer, but well-described phenomenon of congenital partial aplasia of the posterior arch of the atlas has also been described.[32] In this condition, a bony defect of the posterior arch of C1 is replaced with a dense fibrous band that is mobile and impinges the posterior spinal cord.

Diagnostic evaluation of these patients should begin with plain radiographs, including dynamic flexion/extension views. If the patient has myelopathic symptoms, then a CT scan should be performed to identify any intrusion into the spinal canal. Finally, an MRI should be obtained to assess the spinal cord itself. Sagittal CT and MRI images can help evaluate the anteroposterior (AP) dimension of the spinal canal. It is generally accepted that an AP diameter of 13 mm or less may be associated with neurologic compromise.[4]

Treatment of occipitalization of the atlas is variable. In the case of a fused anterior arch of C1 to the occiput without associated translation of C1 relative to C2 (which may indicate an incompetent transverse ligament), the posterior elements of C1 may be resected. Associated instability either atlantooccipital or atlantoaxial should be treated with internal fixation and fusion. The first step is to determine whether atlantoaxial subluxation or basilar invagination is reducible by traction. In general, the younger the child, the more likely the lesion is reducible.[33] If reduction is inadequate to reduce the spinal cord compression, as seen on MRI, ventral decompression is required followed by fusion and stabilization. Techniques for these particular scenarios are discussed elsewhere in the text.

ACHONDROPLASIA

Achondroplasia deserves special mention because of its significant association with craniocervical deformities. Although most cases are sporadic, a familial form is transmitted in an autosomal-dominant fashion and is linked to the fibroblast growth factor receptor-3 gene.[34] Neurologic symptoms including irregular respiratory patterns may be seen in up to 85% of achondroplastic newborns.[35,36] The rate of sudden infant death is significantly higher than that of the general population.[37] A narrowed foramen magnum and upper cervical stenosis may be seen in a majority of patients with CT imaging.[38,39] The rate of growth of the foramen magnum is slowed during the first year of life, and this results in compression at the cervicomedullary junction (Fig. 44-4).[40] Thus, the treatment for these patients includes suboccipital decompression and duraplasty to accommodate the lower brainstem and upper spinal cord. Subaxial cervical stenosis has also been

Fig. 44-4 Compression at the craniocervical junction in achondroplasia. The *white arrow* indicates the area of compression of the cervicomedullary junction with signal changes in the spinal cord in this T2-weighted magnetic resonance image.

associated with achondroplasia and is usually treated with laminectomy.[41]

BASILAR INVAGINATION

This condition is characterized by the encroachment of the foramen magnum by the upper cervical spine, usually the odontoid process. The lower brainstem may be severely impacted by the dens as it is positioned aberrantly through the foramen magnum and into the posterior fossa. Primary or true congenital basilar invagination is associated with other abnormalities including atlantooccipital fusion, hypoplasia of the atlas, odontoid abnormalities, Klipped-Feil Syndrome (KFS), and achondroplasia. Condylar hypoplasia elevates the position of the axis and atlas and often leads to basilar invagination. Acquired basilar invagination, or basilar impression, is caused by softening of the bone at the base of the skull resulting from degenerative disorders such as Paget's disease of bone, osteogenesis imperfecta, Hurler's syndrome, and severe rheumatoid or osteoarthritis, tumors, or infection.

The etiology of primary basilar invagination may be the result of an insufficient amount of paraxial mesoderm leading to underdevelopment of the occipital somites, subsequent shortening of the clivus, and an enlargement of the foramen magnum in the AP dimension.[42] During chondrification of the odontoid process, the cartilaginous dens may transiently reach into the foramen magnum.[43] Normally, the odontoid process will descend to its normal position below the foramen magnum in the fetal period, but if this is incomplete, then basilar invagination may result.

Generally, basilar invagination may be defined by the amount of protrusion of the odontoid process through the foramen magnum. A general rule of thumb is that basilar invagination should be suspected if the C1-C2 facet complex cannot be adequately visualized on a normal open-mouth anteroposterior view of the upper cervical spine. Several methods can diagnose basilar invagination. Common methods include Chamberlain's, McRae's, or McGregor's lines (Fig. 44-5). All are based on lateral radiographs of the cervical spine. Chamberlain's line is drawn between the opisthion to the posterior aspect of the hard palate. McGregor's line is drawn from the posterior aspect of the hard palate to the base of the foramen magnum. McRae's line is drawn from the anterior to the posterior rim of the foramen magnum. Because McRae's line defines the opening of the foramen magnum, an odontoid process that projects above this line is likely to induce symptoms. McRae reported that a reduction of the opening of the foramen magnum to less than 19 mm was likely to produce neurologic deficits.[27] Although plain radiographs have their utility as screening methods, the best imaging modality is MRI. MRI provides clinically useful information such as the degree of impingement of neural structures.

Children with basilar invagination often present with a short neck and a limited, painful range of motion. Basilar invagination has recently been separated into two distinct groups.[44] One group had only the radiographic finding of basilar invagination; the other group had an associated Chiari I malformation. Symptoms are highly variable and often do not become apparent until the second or third decade of life. Symptoms may also be elicited by minor trauma. Patients may often present with muscle weakness, neck pain, posterior column dysfunction, and paresthesias.[44] Common presenting signs include localized torticollis, limited neck mobility, low hairline, webbed, and short neck.[44]

Treatment of basilar invagination usually begins with traction. This maneuver can reduce the compression of the neural structures by the odontoid (Fig. 44-6). Once the impression is reduced, a posterior occipitocervical stabilization procedure can be performed to maintain the reduction. In the instance that the invagination cannot be reduced, a transoral decompression may be required, followed by a posterior occipitocervical fusion. Patients with an associated Chiari decompression may benefit from foramen magnum decompression with duraplasty, and few will require a stabilization procedure.[44]

INIENCEPHALY

Iniencephaly consists of congenital cervical synostoses, fixed retroflexion or hyperextension of the head, severe cervical lordosis, and incomplete posterior neural closure (occipital

1	Wackenheim's clivus canal line	4	McGregor's line
2	Chamberlain's line	5	Height index of Klaus
3	McRae's line	6	Spinous interlaminar line (posterior canal line)
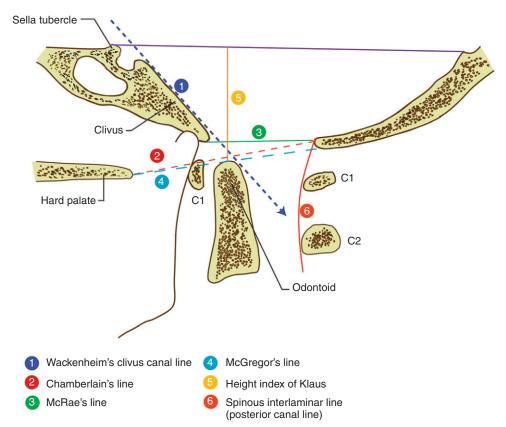
Fig. 44-5 The various methods (craniometry) used to assess basilar invagination including Wackenheim's line, Chamberlain's line, McRae's line, McGregor's line, height index of Klaus, and the spinous interlaminar line.

Fig. 44-6 Magnetic resonance image showing severe compression of the brainstem from basilar invagination.

defect and/or cervical spina bifida).[45–47] Many patients now survive into adulthood but are often incapacitated by cervical lordosis and hyperextension of the head. Treatment of this condition has included suboccipital release, gradual flexion of the head over several weeks in a halo brace, and occipitocervical fusion to maintain the correction.[48]

ANOMALIES OF THE AXIS AND OS ODONTOIDEUM

Congenital abnormalities of the second cervical vertebra often involve some malformation of the odontoid process. The degree to which the odontoid process may be affected ranges from hypoplasia to complete aplasia.[4,49] The resultant clinical picture may be one of atlantoaxial instability because of the fact that the normal anatomic and biomechanical complex involving the transverse cruciate ligament and an intact odontoid process are not present. The true incidence of anomalies of the axis is unknown, but they are seen in association with Down syndrome, Morquio syndrome, and a variety of other skeletal dysplasias.

Os odontoideum is described as dissociation between the body of C2 and the dens such that a disconnected ossicle takes the place of an intact odontoid process (Fig. 44-7). This should be differentiated from *ossiculum terminale persistens,* a condition whereby the very tip of the dens, the ossiculum

Fig. 44-7 Os odontoideum. Lateral radiograph showing disconnection of the dens from the body of C2. The *white arrow* indicates the disconnected ossicle.
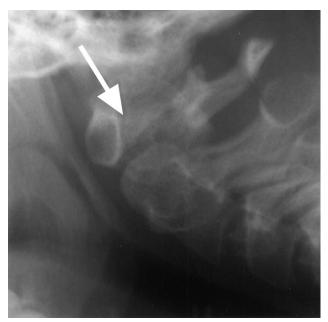
terminale, fails to fuse with the remainder of the dens. The ossiculum terminale usually is firmly bound to the main body of the dens by cartilage and consequently is seldom the source of instability. Historically, controversy existed regarding the etiology of os odontoideum. Some authors favored a congenital cause, whereas others postulated that minor trauma in early childhood resulted in disruption of the vascular supply of the developing dens causing it to dissociate from the axis.[50–52] Currently, most authors favor the latter explanation, as a chronic nonunited fracture of the odontoid process.

Patients with atlantoaxial instability from congenital malformations of the odontoid are at risk for significant neurologic injury. Many patients present with neck pain and headache from the instability, but instances of myelopathy and transient quadriparesis from minor trauma have been reported. If instability is suspected, then dynamic imaging with flexion/extension films should be obtained. MRI should also be obtained to evaluate the spinal cord. Treatment for atlantoaxial instability resulting from os odontoideum or a maldeveloped odontoid process usually requires posterior stabilization. The highest success rates have been achieved with a C1-C2 transarticular screw fixation method.[53] Some authors have also argued that even in the setting of asymptomatic instability, patients should undergo a fusion procedure as even minor trauma may result in a significant neurologic injury.[54]

KLIPPEL-FEIL SYNDROME

Perhaps the most commonly encountered congenital malformation of the cervical spine is Klipped-Feil Syndrome (KFS). In 1912 Klippel and Feil reported the case of a patient with a short neck, low hairline, and limited neck mobility[55] (Fig. 44-8). At autopsy, the patient was found to have a spine consisting of only 12 discernible vertebrae. Although the triad described previously is considered classic for the syndrome, it is now recognized that fewer than 50% of patients with congenital fusion of the cervical spine will have all three signs.[24] KFS refers to any congenital fusion of the cervical spine, with or without the classic triad and is now commonly diagnosed in those with a congenital fusion of two or more cervical vertebrae (Fig. 44-9).[56] The true incidence is impossible to know, but it is estimated that it occurs in approximately 1 in 40,000 to 1 in 42,000 births.[24,57] Several other associated abnormalities are seen with KFS including congenital scoliosis (Fig. 44-10), rib abnormalities, deafness, genitourinary abnormalities, Sprengel's deformity, synkinesia, cervical ribs, and cardiovascular abnormalities.[23,58–60]

The etiology of KFS is complicated by the variable clinical presentation. In general, the syndrome is felt to result from the mutation or aberrant expression of genes involved in sclerotomal segementation.[61,62] At least three distinct genetic forms of KFS have been described in the literature, each with associated birth defects.[24] An association with a mutation in the fibroblast growth factor receptor-3 gene in patients with autosomal dominant coronal synostosis, dysmorphic vertebrae and ribs, and Sprengel's deformity has been described.[63] However, a similar phenotype has been observed without this particular mutation.[64] Recent pedigree analysis found that KFS and vocal cord impairment segregated with a paracentric inversion of chromosome 8q. The genetic locus on this chromosome was identified as *SGM1,* which correlates to the *Drosophila* segment polarity gene product engrailed. Other gene families have been implicated in KFS including *Notch, Pax,* and *Hox. Notch* genes regulate embryonic patterning and mutations in some of these genes in mice

Fig. 44-8 Photograph of a patient with Klippel-Feil syndrome. Note the short, webbed neck, and low-lying hairline.

Fig. 44-9 Lateral radiograph of the cervical spine showing multiple cervical fusions in Klippel-Feil syndrome.

Fig. 44-10 Radiograph showing scoliosis of the thoracic spine, the most common finding associated with Klippel-Feil syndrome.

result in disrupted somite segmentation.[65,66] Mutations in the *Notch* gene pathway have yet to be linked to KFS, however. *Pax* and *Hox* genes, particularly *Pax1, Hoxd3,* and *Hoxd4,* are also important for somitic development and segmentation.[67,68] Disruption of selected genes in each of these pathways has shown to result in cervical spine malformations in mice, but have not correlated with congenital cervical fusions in humans.

KFS has been characterized by several different classification schemes. In 1919 Feil divided the syndrome into three types based on the site and extent of the congenital fusion. Type I described fusion of cervical and upper thoracic vertebra. Type II described patients with only one or two cervical fusions. Type III included patients with fused cervical vertebra as well as lower thoracic or lumbar fusions. Some clinical correlations have been made with each type. Types I and III have shown a tendency toward scoliosis, and are typically inherited in an autosomal recessive manner.[57,64,69,70] Type II (the most common type) has been associated with other skeletal abnormalities, such as Sprengel's deformity and is generally inherited in an autosomal-dominant fashion.[64,69,70] In general, this classification scheme has not proved clinically useful. A second scheme was used to describe three patterns of potentially unstable fusions.[59,71] Type I is characterized by fusion of the C2 and C3 with occipitalization of the atlas. Type II describes a long cervical fusion with an abnormal craniocervical junction. Type III describes two segments of block fusion separated by a single nonfused interspace.

Type III was felt to be the most susceptible to instability resulting from the theoretical strains on the nonfused interspace, but long-term clinical data have not confirmed this. Others have correlated various KFS deformities with dynamic imaging to create a classification scheme.[72] These authors showed that patients with hypermobility at the upper cervical spine were at greatest risk of neurologic sequelae, whereas patients with hypermobility of the lower cervical spine were at higher risk for progressive degenerative disk disease. The most recent classification (reviewed by Tracy et al.[24]) separates KFS into four classes and incorporates the mode of inheritance.[73] Fusions at C1 with or without associated caudal fusions are Klippel-Feil 1 (KF1) with an autosomal-recessive inheritance pattern. KF1 is most often associated with other anomalies. KF2 describes patients with fusions no higher than C2-C3. These patients have an autosomal-dominant inheritance pattern and 100% penetrance of the C2-C3 fusion. KF3 is defined as congenital fusions caudal to C2-C3 and has either autosomal-recessive

or dominant inheritance. KF4 is described as synonymous with Wildervanck syndrome which is characterized by congenital cervical fusion, congenital hearing loss, and the Duane anomaly. It is thought to have an X-linked dominant inheritance pattern and is hemizygous lethal.

As mentioned previously, the classic triad of KFS is seen in fewer than 50% of patients. The most common clinical presentation of KFS is limited range of motion, particularly lateral bending. If fewer than three cervical vertebrae are fused, however, motion of the cervical spine may appear normal as adjacent levels may compensate. Thus patients with extensive neck fusions may present at an earlier age. Also, higher fusions near the craniovertebral junction present earlier (Fig. 44-11)—C1-C2 fusions often present with pain in childhood whereas lower cervical fusion present in the second or third decade of life or later, when symptomatic junctional degeneration develops.[74] Torticollis or neck webbing is seen in only 20% of patients with KFS.[59,75–77] Torticollis is not specific to any particular cervical spine anomaly. It may be a manifestation of a muscular condition or bony abnormalities.[78] Hemivertebra of the atlas is often seen in association with torticollis. Other conditions are associated with torticollis as well including posterior fossa tumors, infections, and cervicothoracic scoliosis. In patients with severely limited neck mobility and a low posterior hairline, iniencephaly should be suspected (see previous).

Fig. 44-11 Sagittal reconstruction CT scan showing congential fusion of C2 and C3 in a young patient with Klippel-Feil syndrome.

Several other clinical presentations are associated with KFS. Facial asymmetry, such as that seen in the congenital craniosynostosis syndromes of Crouzon and Apert, may be associated with cervical spine anomalies.[79] Other associated syndromes include Wildervanck, Rokitansky-Kuster-Hauser, or Goldenhar. Hearing loss can be present in up to 30% of patients with KFS, and hearing should be evaluated formally if the diagnosis is suspected.[76,77] Deafness may be the result of ankylosis of the ossicles, footplate fixation, absent external auditory canal, or sensorineural hearing loss.[80,81]

Numerous musculoskeletal anomalies are associated with KFS, the most common being scoliosis (usually congenital), occurring in up to 60% of patients. Sprengel's deformity, a congenital elevation of the scapula, can be seen in 20% to 35% of patients with KFS. It is thought to arise from failure of descent of the scapula from the first embryologic cervical level to its normal position, just caudal to the first rib.[82] The time of descent coincides with cervical somite resegmentation implying a connection between an anomalous scapular descent and aberrant fusion of cervical vertebral bodies. Other musculoskeletal anomalies include cervical ribs, rib anomalies, and hemivertebrae.

Cardiovascular abnormalities are reported to occur in up to 14% of patients with KFS. A ventricular septal defect, either independently or in combination with other cardiac defects, is the most common associated abnormality.[83,84] Some authors recommend a baseline echocardiogram, especially for those patients undergoing surgery.

Various neurologic disturbances may be seen in patients with KFS. These include developmental abnormalities of the central nervous system (CNS) such as brainstem malformations, myelopathy as a result of long-standing spinal cord compression, radiculopathy as a result of nerve root irritation, and nonspecific symptoms of headache, weakness, and numbness. An unusual, but not uncommon, associated neurologic sign is synkinesis. This is a condition in which involuntary mirrored motions, primarily in the upper extremities, are observed. Although it may be seen in normally developed children, up to 20% of patients with KFS will exhibit the condition.[85,86] Although its etiology is unknown, autopsy results of two patients with KFS and synkinesia showed an incomplete pyramidal decussation. Synkinesia is generally effectively treated with occupational therapy, and the condition often subsides as the patient ages.

Genitourinary abnormalities are also associated with KFS. Up to 64% of patients will have some defect in the genitourinary pathway, with the most common manifestation being unilateral renal agenesis.[59,87,88] Developmentally, the renal system begins its development at 28 to 30 days of embryologic life. An insult to the developing embryo between the fourth and eighth week of life may result in anomalies of the genitourinary system as well as those related to resegmentation of cervical somites. Other renal anomalies including malrotation of the kidney, absence of both kidneys, ureteral agenesis, hydronephrosis, and a variety of malformations of

the kidney.[89–91] Patients with KFS should receive an ultrasound of the renal system. Intravenous pyelography is suggested for patients with an abnormality detected on ultrasound, or an inconclusive ultrasound study.[87]

Abnormalities noted in the renal system may point to abnormalities of the reproductive system, particularly in females. An absent vagina, uterus, and/or ovaries have all been reported in association with KFS.[89,92,93] Male reproductive abnormalities have also been reported including dysplastic or undescended testes. In general, if abnormalities of the renal system are identified, a thorough ultrasonic examination of the reproductive system should be performed as well.

Imaging of the cervical spine is important for an accurate diagnosis. Routine plain radiography consisting of AP, lateral and open-mouth odontoid views are useful initial studies as they can quickly identify an obvious congenital fusion or cervical stenosis. Flexion and extension views provide a dynamic snapshot of the cervical spine and can identify stability of the atlanto-occipital, atlantoaxial, and subaxial joints. Radiography of the cartilaginous spine, seen in children younger than 8 years, can be difficult to interpret. Patients with antero or retrolisthesis of cervical vertebral bodies may have instability, although pseudosubluxation of C2 on C3 or C3 on C4 is physiologically normal in children.[94–96] Juvenile rheu-

matoid arthritis can mimic KFS, although additional clinical information easily distinguishes the two conditions.[57]

MRI should be used in the setting of suspected compromise of the brainstem or spinal cord. Sagittal MRI will allow for a determination of the AP dimension of the spinal canal. Additionally, MRI allows for detection of other CNS lesions such as syringomyelia, tethered cord, diastematomyelia, and Chiari malformation.[24,97] In addition to the imaging modalities that are useful for screening for associated systemic abnormalities (e.g., renal ultrasound, see above), further imaging of the thoracic and lumbar spine is warranted in patients with KFS as they may have abnormalities in these regions such as scoliosis.[57]

Treatment for KFS is dependent on the nature of the pathology. Because many patients are asymptomatic throughout life, treatment must be individualized. Patients who are asymptomatic, have stable fusion patterns, and adequate spinal canal diameter may never develop symptoms.[56,98] Although many patients present with minor symptoms, it should be recognized that minor traumatic events have resulted in catastrophic spinal cord injuries resulting in tetraplegia or even death.[98–108] For patients with atlanto-occipital instability, a technique for occipitocervical fusion should be used. Atlantoaxial instability is best approached with C1-C2
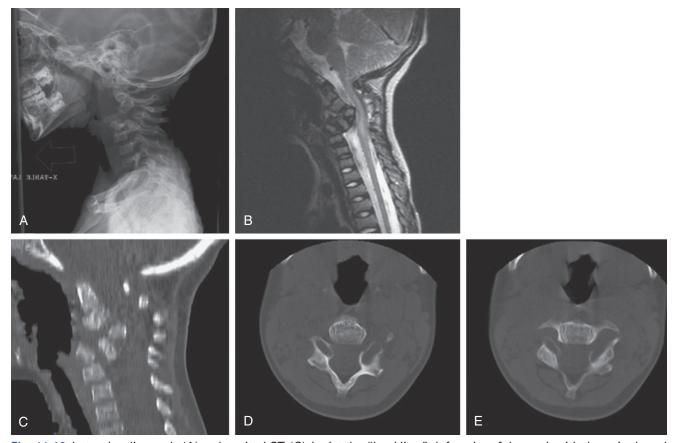

Fig. 44-12 Lateral radiograph *(A)* and sagittal CT *(C)* depict the "buckling" deformity of the neck with the spinal cord being compressed ventrally as shown in the sagittal MRI *(B).* The absence of an osseous bridge between the anterior and posterior elements and the enlarged and abnormally shaped foramen transversarium are seen in the axial CT *(D, E).*

transarticular screw fixation techniques. The operative details for these techniques are described elsewhere in the text. Patients with subaxial instability typically will not present with neurologic symptoms, but may have significant degenerative disk disease. These patients may be successfully treated with diskectomy and fusion. Cervical stenosis is generally treated with posterior decompression and fusion if necessary.[109,110]

MISCELLANEOUS DISORDERS

Cervical spondylolysis is a rare congenital spinal anomaly. It is defined as a cleft between the superior and inferior articular facets of the articular pillar or lateral mass, the cervical equivalent of the pars interarticularis of the lumbar spine.[111,112] Previously, cervical spondylolysis has only been described in adults.[113,114] Characteristic radiographic findings include well-corticated margins at the defect, a characteristic "bow-tie" deformity, and ipsilateral dysplastic facets. Compensatory hypertrophic changes of the adjacent articular processes, spina bifida, and spondylolisthesis are often, but not always, seen.[115] Cervical spondylolysis most commonly occurs at a single level (the most common level is C6), but several cases of multilevel involvement have been reported.[112,116–119]

Recently, an osseous disconnection between the anterior and posterior elements resulting in a severe kyphotic deformity and myelopathy has been described[120] (Fig. 44-12). This has been described as congenital multilevel cervical disconnection syndrome. Patients with this disorder were treated with extensive anterior and posterior reduction, decompression, reconstruction and stabilization/fusion procedures. It is hypothesized that this anomaly is a result of a failure of connecting chondrification centers to form (Fig. 44-13).
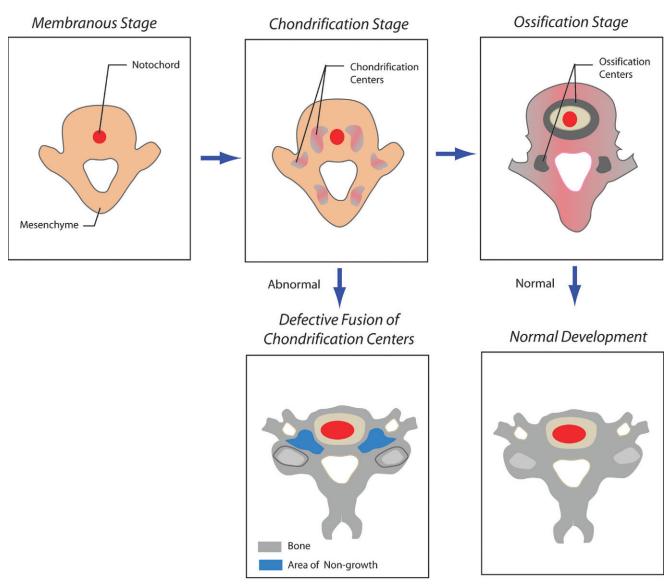

Fig. 44-13 The normal chondrification and ossification stages of spinal embryogenesis. The anomaly is due to improper fusion of chondrification or ossification centers.

CONCLUSION

Developmental anomalies of the cervical spine vary widely. The development of the cervical spine is dependent on the interaction of several complicated genetic pathways. An alteration of these pathways may result in the malformation of the cervical spine, many of which are due to a failure of segmentation of cervical somites. It is important to recognize that some of these malformations may be associated with other defects involving the cardiovascular, neurologic, renal, and reproductive systems.

The true incidence of these disparate anomalies is not known for certain, partly because of their frequent asymptomatic nature. Identifying the symptomatic anomalies requires adequate imaging. Patients may present with abnormalities as simple as two congenitally fused vertebrae requiring no treatment, or as complex as craniocervical instability requiring occipitocervical fusion. Recognizing those congenital abnormalities that contribute to an unstable cervical spine or critical spinal stenosis may prevent a catastrophic spinal cord injury.

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Spinal Biomechanics of the Pediatric Spine: Clinical Applications

The pediatric spine is a distinct entity with little biomechanical analogy to the adult spine. An understanding of the unique biomechanical principles and properties of the pediatric spine is requisite of the treating clinician. These principles and the changes that occur with age are particularly relevant to operative and nonoperative decision making in the management of pediatric spinal trauma, instability, and deformity. Successful management of the traumatized pediatric spine is predicated on a thorough understanding of and application of the principles and characteristics of the immature spine. It is clear that our collective knowledge of pediatric spinal biomechanics and the biologic basis for these characteristics remains incomplete. Nonetheless, treatment of pediatric spinal injury is guided by application of our current knowledge base. This chapter will focus on the clinical application of these biomechanical characteristics of the immature spine with emphasis on assessment and management of spinal instability.

CHARACTERISTICS OF THE PEDIATRIC SPINE

There are several well-described characteristic differences between the pediatric and adult spine. A fundamental difference is the continued potential for growth in immature musculo skeletal tissues. The presence of physes is another unique property with particular relevance to spinal injury. In addition, the pediatric spine has more resting elasticity or malleability than does the adult counterpart. The pediatric spine responds to applied stress with a more robust response than do adult tissues. The culmination of these and other unique biomechanical characteristics of the pediatric spine coupled with the unforgiving, inelastic properties of neural tissue increases the complexity of management of pediatric spinal injury.

ANATOMIC DIFFERENCES

There are several important anatomic considerations that ultimately affect spinal function and the ability of the spine to resist loads. The vertebral bodies and intervertebral disks possess structural differences from the adult spine, which partially account for the robust capacity to remodel and grow. The most obvious unique anatomic characteristic is the presence of the physes that contribute the capacity for growth. As discussed later, the thick cartilaginous endplates of the immature vertebral bodies can be a source of injury, analogous to physeal injuries in the appendicular skeleton. The ligaments, intervertebral disks, and surrounding soft tissues have increased elasticity, whereas the supporting musculature is less well developed. Pediatric bone compared with adult bone demonstrates a lower modulus of elasticity and lower strength with increased energy absorption and deformation before failure.[1,2] This combination allows increased resilience to injury of osseous structures. The child's spinal column can be stretched by 2 cm, whereas the encased neural structures can stretch only 5 to 6 mm before significant cord injury.[3] The intervertebral disks also change with maturity and age. The immature nucleus pulposus has a larger water content, which allows more shock absorption. According to the work of Keyes and Compere[4] in 1932, the water content of the nucleus pulposus decreases from 88% at birth to 80% at age 12 and to 70% at age 70. This desiccation, combined with increased collagen cross linkage, leads to a loss of elasticity with aging and thus the corresponding change in injury pattern to that of an adult.

In the immature spine, the vertebral bodies are wedge shaped, the articular facets are angled more horizontally, the endplates are cartilaginous, and there is increased elasticity and laxity to the interspinous ligaments.[5] The facet joints in the upper cervical spine evolve from a quite horizontal angle of 30 degrees at birth to 60 to 70 degrees by adolescence. The facet joints in the lower cervical spine changes from 55 to 70 degrees (Fig. 45-1).[6] This change in orientation may contribute to the occurrence of pseudo-subluxation and the relative increased incidence of upper cervical spine injuries in the very young child. The joints of Luschka, which do not ossify until around 7 years of
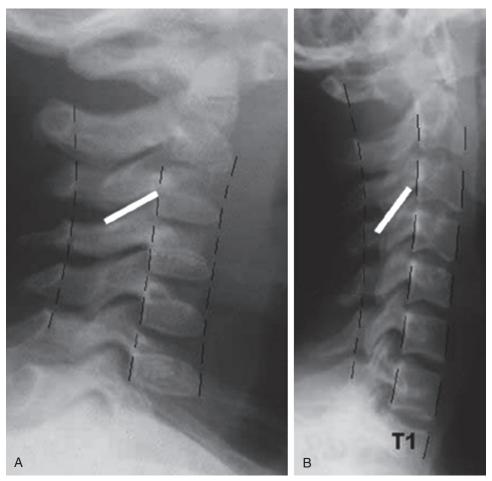
Fig. 45-1 The *solid white line* represents the more horizontal orientation of the facet joints in the young child *(A)*. The *dashed black lines* indicate the spinolaminar line, the posterior vertebral body, and anterior vertebral body lines. The *solid white line* demonstrates the more vertical orientation of the facet joints in the older child/adolescent *(B)*.

age, may also contribute to increased stability with increased age.[7] An additional anatomic consideration is the high ratio of the weight of the young child's head compared with that of the older child (Fig. 45-2). The large head to body ratio in the younger than 8 years population cause increased torque and acceleration stress in the upper cervical spine.[8] With age, this ratio decreases, and the biomechanical result of this phenomenon is that the fulcrum in spinal flexion shifts from C2-C3 to C5-C6 by late childhood.[8,9] By 8 years of age, radiographic spinal anatomy is similar to that of the adult, as are the patterns of spinal injury.[10] Anatomic differences such as relatively larger heads, less developed neck musculature, and relative ligamentous laxity make children younger than 8 years more vulnerable to subluxation and distraction type spinal injuries.[5,11,12] In the older pediatric population (aged 10 to 16 years), the anatomic, biomechanic, and injury patterns become more analogous to those of adults. Specifically, the vertebrae become more rectangular, losing the wedge shape, the orientation of the facets becomes more vertical and the ligamentous laxity decreases. Consequently,

there are fewer fractures in the younger than 10 years population when compared with the 10 to 16 years population in whom the vertebrae have further ossified.[13,14]

GROWTH AND GROWTH PLATES

Arguably the most important concept in pediatric spinal instability and deformity is that of growth. Growth is important for two major reasons. The first is that the concept of growth remaining must be understood when managing traumatic injuries and predicting progression of deformity. This unique capacity of the immature spine makes management of injury, instability, and deformity much more complex. A second reason growth is important is for understanding the anatomic and biomechanical properties rendered by the focus of growth, the physis. The growth plate is known to be the weakest link in the axial skeleton when subjected to tensile forces. A clinical example of this phenomenon is the odontoid fracture in the skeletally immature. The normal physis of the odontoid process is transverse and occurs at approximately the level of an adult
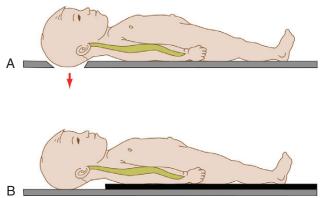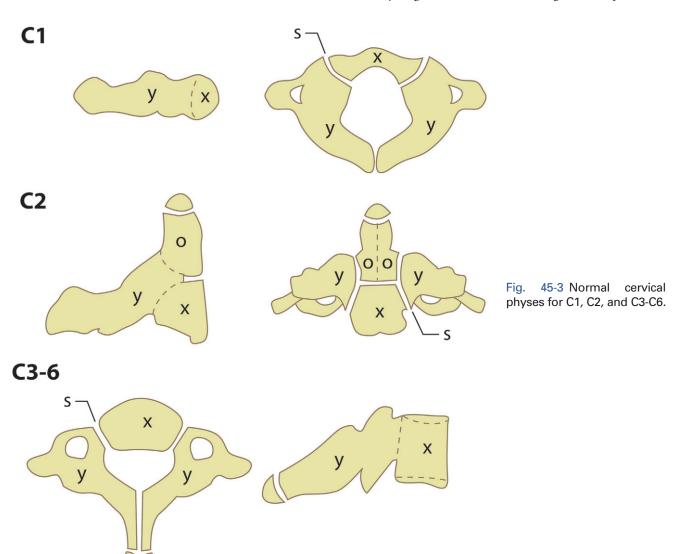
Fig. 45-2 The young child has a proportionally larger head to body ratio. When immobilizing the young child this must be taken into consideration to allow the spine to be immobilized in a neutral position. This is accomplished by either a "cutout" in the immobilization board *(A)* or elevating the rest of the body on a flat spine board *(B).*

type II odontoid fracture (Fig. 45-3). Unlike the adult situation, the injury occurs through a physis and the healing potential is accordingly tremendous, making nonunion exceedingly rare.[15]

Injuries to the ring apophysis (Fig. 45-4) may occur in association with lumbar disk herniations. As part of the annulus protrudes from the disk space, traction is pulled on the ring apophysis. This results in an avulsion of a fragment of the endplate and the radiographic appearance of an extruded fragment (Fig. 45-5).[16] Although injuries to the anterior column are clearly affected by the presence of a physis, the posterior column is not characterized by the presence of similar physes. The result is that the posterior column is characterized by bony fractures and ligamentous injuries in the skeletally immature.

From a biomechanical standpoint, growth is important because it applies a force to the spine. The effect of this force depends on the magnitude and direction of the applied load. The magnitude of this force depends on the velocity of growth. The force is thus greatest in periods of

Fig. 45-3 Normal cervical physes for C1, C2, and C3-C6.

Fig. 45-4. Normal ring apophysis of C7 in a 12-year-old child. The ring apophysis represents a secondary ossification center and contributes minimally to longitudinal growth.

Vertebral body

End plate

Intervertebral disc

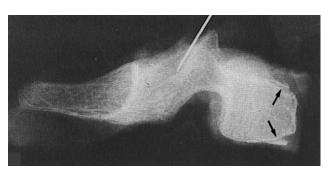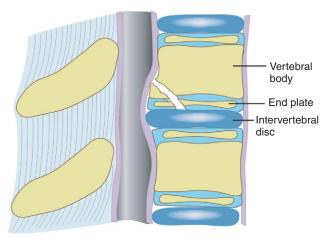Fig. 45-5 Diagram demonstrates an avulsion fracture of the ring apophysis. The intervertebral disk and vertebral end plates are labeled.

highest growth velocity such as infancy and adolescence. These growth-mediated forces may significantly change spinal structure because they are applied over long periods of time. In the normal growing child, these forces are coordinated in each plane and result in longitudinal growth. In congenital formation or segmentation failures and in surgical arthrodesis, the loss of balance of these forces may result in progressive deformity.[17,18] The crankshaft phenomenon, which may occur in posterior-only arthrodesis of the growing spine, is an example of the complexity added to the management of spinal deformity in the child's spine. This phenomenon may occur despite successful posterior arthrodesis because there persists some plasticity in the fusion mass.[19] Anterior and posterior arthrodesis is thus often recommended to avoid the crankshaft phenomenon in children younger than 10 years with open triradiate cartilage and Risser stage 0.[20–22]

ADAPTABILITY, MALLEABILITY, AND MOBILITY

Spinal development and the maintenance of spinal stability is a complex orchestration of biochemical, biomechanical, and cellular processes. The shared goals are maintenance of spinal stability and mobility. Normal spinal function involves resisting and adapting to deforming forces. The capacity of the pediatric spine to adapt to an applied load far exceeds that of the adult spine. Adaptability is an active process characterized by bony remodeling in response to an applied load. This is a result of the capacity for growth, the capacity to remodel, and the lower modulus of elasticity compared with the adult spine.[23,24] The adaptability of the pediatric spine can be exemplified by observing the preservation of sagittal balance, affected by a compensatory lumbar curve in response to a thoracic curve in idiopathic scoliosis. The development of this secondary curve maintains sagittal and coronal alignment and is thought to develop in response to asymmetric loading of the spine. Although musculoskeletal tissues can adapt, neurologic tissues lack this unique property, even in the immature child. As a result, the child may be quite functional with severe skeletal deformity; however, excessive stress placed on neural tissues may result in neurologic compromise.

The concept of malleability describes the passive process whereby the pediatric spine is able to deform in response to an applied load without failure of the osseoligamentous restraining structures. This is a function of inherent elasticity of the ligaments and the low modulus of elasticity of immature bone. Nuckley et al.[24] demonstrated in baboon spines that the modulus of elasticity, yield strength, stiffness, and volumetric bone mineral density increase with age. Malleability of the spine decreases with age. The pediatric spine is therefore able to absorb considerable energy prior to osseous failure. These characteristics partially explain why spinal fractures are significantly less common in children. As mentioned previously, neural tissues lack this capacity such that spinal cord injury without radiographic abnormality (SCIWORA) can occur because of vertebral overdistraction or subluxation while the osseoligamentous structures remain intact.

As is true in most regions of the child's musculoskeletal anatomy, there is a greater physiologic range of motion in the pediatric spine. This increased mobility results from the greater laxity of ligamentous tissue as well as the more horizontal orientation of the facets joints.[25] Instability at the atlantoaxial joint is identified by an atlanto-dens interval (ADI) greater than 4 mm in children, compared with 3 mm in adults.[26] This difference is secondary to the combination of a portion of the dens and atlas being unossified and increased hypermobility resulting from physiologic ligamentous laxity (Fig. 45-6). Another example of physiologic hypermobility is pseudosubluxation of C2 on C3. In pseudosubluxation, a normal variant, the physiologic hypermobility does not cause a pathologic condition under
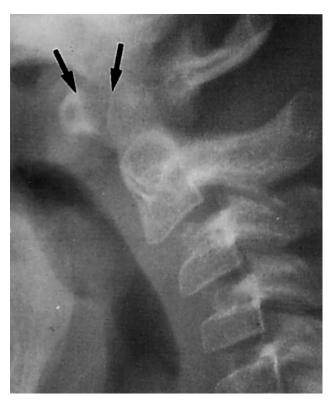
Fig. 45-6 Normal lateral upper cervical radiograph in a child in flexion. The atlanto-dens interval (ADI) in the normal child is 4 mm or less. In children the interval has a "V" shape in contrast to the shape of the ADI in adults *(black arrows).* The anterior tubercle of C1 also rides quite cephalad on the dens. This is the normal pattern.

Fig. 45-7 The spinolaminar line of Swischuk is indicated by the *second solid white* line from the left. This patient has pseudosubluxation of C2 on C3. This is confirmed by the smooth contour of Swischuk's spinolaminar line.

physiologic load.[27] Pseudosubluxation of C2 on C3 has been demonstrated to be a normal variant exemplifying hypermobility and may be regarded as such even in the polytrauma situation (Fig. 45-7).[28] A pathologic state is identified by greater than 4 mm of subluxation, prevertebral soft tissue swelling, loss of alignment of the facet joints, and a noncontinuous spinolaminar line of Swischuk. Whereas the adolescent population is more susceptible to cervical injuries in the lower cervical spine, these features predispose younger children to upper cervical spine injuries, SCIWORA, and ligamentous injuries (Fig. 45-8).[5,29]

SCOLIOSIS

In scoliosis, spinal deformity occurs in several planes. Often the most clinically evident is the coronal plane deformity. However, the importance of the noncoronal plane deformities in both etiology and management has become more clear in recent years. An understanding of the relevant biomechanical principles is important for the study of the initiation, progression, and management of idiopathic scoliosis. Many theories on the etiology of idiopathic scoliosis have been proposed. Most of these have since been disproved, and

despite intense research efforts, there continues to be no clear consensus on how this complex process occurs. There does appear to be an equilibrium or postural abnormality in adolescents with idiopathic scoliosis compared with controls, although the significance of this in initiation, progression, or management of AIS remains unknown.[30–32] Machida et al.,[33] in 1995, suggested that melatonin may play a role in initiation of AIS. Using a validated model for producing scoliosis, they treated pinealectomized chickens with melatonin, serotonin, or no treatment and found a lower incidence and less severe scoliosis in the melatonin group. A later study noted decreased melatonin levels in adolescents who had curves which progressed more than 10 degrees in 12 months compared with age-matched controls who progressed less rapidly.[34] Hilibrand et al.,[35] however, found no correlation when they compared melatonin levels in a population of children with scoliosis to those in a cohort of unaffected controls. The mechanism by which pinealectomy causes scoliosis remains a matter of debate. It is likely that the disturbance of equilibrium and postural maintenance caused by pinealectomy play a role in altering the normal spinal biomechanics.[33] Abnormalities of postural control and vestibular

Fig. 45-8 Spinal cord injury without radiographic abnormality (SCIWORA). T2-weighted image of the cervical spine in a patient without plain radiographic evidence of injury but clear neurologic deficit. MRI demonstrates cord signal change from C4-C7.

otolithic function have also been implicated in the initiation of scoliosis deformities.[31,36]

Several biomechanical principles have been implicated in the initiation of scoliosis, in addition to muscle imbalance. Loss of thoracic kyphosis has been implicated.[37] White and Panjabi[25] demonstrated that the spine, which is a linked column, loses stability when it is straightened. The clinical application of this concept is the reproducible finding that coronal plane deformities are associated with relative lack of thoracic kyphosis and subsequent rotational deformity. The loss of thoracic kyphosis results in the spine being unstable to rotation. Deacon et al.[38] demonstrated with derotated radiographic views that the lateral radiograph overestimates the amount of thoracic kyphosis in the rotated scoliotic spine. They showed that the routine lateral demonstrated an average of 41 degrees of kyphosis whereas the derotated views revealed a mean apical thoracic lordosis of 14 degrees. This loss of kyphosis may result from the rate of anterior spinal growth exceeding that of posterior spinal growth. The signal

or mechanism behind the initiation and progression of AIS remains unknown. The factors responsible for initiation may even be different from those responsible for progression. It is likely that the initiation and progression of scoliosis deformity is a multifactorial interplay between genetic and biomechanical factors leading to rotation and bending of the spine.

The previously described biomechanical factors also play a role in the progression of deformity. The effects of gravity and asymmetric loading are two important factors in progression of scoliosis. Body weight has been shown through biomechanical models to play a role in progression of scoliosis.[39] Michada et al.[33] showed that in bipedal pinealectomized rats, scoliosis occurred whereas quadruped animals did not develop significant spinal deformity. Although the initiating factor in the spinal deformity of AIS is not understood, it is clear that once deformity occurs, increasing deformity is associated with increasing asymmetry of forces applied to the growth plates. The asymmetric loading placed on vertebral physes results in asymmetric growth. In accordance with the Heuter-Volkman law, vertebrae loaded in compression have inhibited growth compared with those seeing a distractive force.[40] Stokes et al.[41] demonstrated a 110% increased height of hypertrophic chondrocytes in vertebral physes loaded in distraction. In those vertebrae loaded in compression, hypertrophic chondrocyte height was found to be just 53% of control chondrocyte height.

The third major facet of scoliosis is application of biomechanical principles to the management of scoliosis deformities. The primary goal in treatment of scoliosis is the prevention of further deformity. Of course, a secondary objective is to achieve some correction of deformity. There are several other goals that include achieving a balanced spine, pelvis and shoulders, maintaining sagittal balance, maintaining lower lumbar motion segments, and finally cosmetic correction. Prevention of progression is performed by decreasing the deforming forces, by providing counter force, or a combination of these techniques. External devices may be successful in preventing progression or producing relative correction of curves less than 40 degrees. In those curves greater than 40 to 50 degrees, success with external bracing becomes less likely.[42] The forces necessary to correct spinal curvature have been well described. Corrective forces may include longitudinal traction on the concavity, compression on the convexity, and rotational forces. The brace should relatively unload the physes in the concavity of the apex. According to the Heuter-Volkmann principle, this will stimulate growth and structural remodeling of the vertebral bodies.

KYPHOSIS

A review of pediatric spinal biomechanics is incomplete without discussion of thoracic kyphosis. The deformity in kyphosis is limited to a single plane in contrast to scoliosis. Therefore, the complex coupled deformities seen in scoliosis are

not typically encountered in the management of isolated thoracic kyphosis. Although the deformity itself is relatively more straightforward, the propensity toward neurologic compromise and pseudoarthrosis is greater and adds to the complexity of kyphosis prognosis and management. Most cases of neurologic deterioration associated with kyphosis are a direct result of a tethering-type mechanism over the kyphotic segment.[43] Neurologic injury results more commonly from transverse compression than it does from longitudinal distraction. In the case of deformity correction in kyphosis, the combined forces in the transverse plane caused by the deformity and the distractive forces of deformity correction put the thoracic cord at significant risk for neurologic injury.[44] To avoid this, prior anterior decompression may be indicated.

The normal range for thoracic kyphosis is 20 to 50 degrees and can be expected to progress through adolescence until maturation.[45,46] Thoracic kyphosis plays several physiologic roles including maintaining sagittal balance, maximizing thoracic volume, and increasing spinal stability. Cervical and lumbar flexibility can typically compensate for the wide variation seen in the degree of thoracic kyphosis in the younger child. This allows for maintenance of sagittal balance. In the older child, however, as the flexibility of the cervical and lumbar spines decreases, maintenance of sagittal balance is not as forgiving and may result in a clinical presentation.

Kyphosis may occur as a result of several factors including asymmetric growth, loss of the anterior buttress, loss of the posterior tension band, or a combination of these issues.[47] Asymmetric growth, specifically where anterior growth is exceeded by posterior growth may occur as a result of a congenital an acquired condition. The physes of the vertebral bodies are susceptible to damage, which may become a biomechanically significant cause of sagittal spinal deformity resulting from asymmetric growth of physeal chondrocytes. Traumatic spinal injuries may result in physeal injury, which may subsequently progress to a kyphotic deformity.

A second mechanism by which kyphotic deformity may occur is loss of anterior column stability. By definition, this implies an inability to resist displacement of the anterior column under physiologic load. Loss of anterior column stability may occur by acquired or congenital means. Neoplastic, infectious, or congenital failures of formation (Type I congenital kyphosis) may result in progressive deformity.[17,48] In addition a thoracic burst fracture with greater than 50% loss of height is also a risk for progressive kyphosis, particularly in the immature spine.[49] When a kyphotic deformity occurs the center of gravity shifts anteriorly out of sagittal balance. The result of this state of positive sagittal balance then results in an increased lever arm and subsequently more kyphosis.[49]

An additional acquired mechanism by which kyphotic deformity may develop is disruption of the posterior ligamentous restraints. The integrity of these posterior static ligamentous restraints is particularly important in the pediatric spine. This is because of the concept of the malleability of the pediatric spine. The malleability of the pediatric spine allows for rapid progression of deformity. Loss of integrity of the posterior tension band may occur by several mechanisms. Traumatic posterior ligamentous injury from a flexion-distraction type mechanism in the skeletally immature child may result in rapidly progressive thoracic kyphosis. Similarly, thoracic laminectomy in the immature spine portends a significant risk of progressive kyphosis.[50–52] Development of kyphotic deformity is quite predictable with isolated laminectomy and facetectomy in patients younger than 10 years.

A final point on kyphosis is the distinction in the immature spine between flexible or postural kyphosis and more rigid deformity. The rigidity and severity of the deformity dictates whether management can be with bracing, or if a surgical intervention would be indicated. Further, when a surgical procedure is indicated, the extent of the procedure is dictated by the degree of deformity and rigidity. Because the broad normal range of thoracic kyphosis ranges up to 50 degrees, curves that could be actively corrected to less than this degree would likely be manageable with bracing. In contrast those curves that could not be reduced to less than 60 degrees are less likely to have a favorable result.[53] Those more flexible severe curves that can be corrected to 55 degrees can likely be managed with a posterior-only instrumented procedure.[54] In contrast, less correctable deformities would warrant an anterior release combined with the posterior instrumentation. Winter et al.[44] found a pseudoarthrosis rate of 55% with posterior only procedure compared with a pseudoarthrosis rate of 15% in anterior/posterior procedures.

SPONDYLOLYSIS AND SPONDYLOLISTHESIS

The study of spondylolisthesis is the study of forward slippage of one vertebral body on a caudal vertebra. This disorder is thus a combination of spinal instability and deformity. In those cases of spondylolisthesis in which there is progression, instability is clearly present. Biomechanic principles are of importance in understanding the management and progression of spondylolisthesis. The incidence of isthmic spondylolysis is 4.4% in children at the age of 6 years and increased to 6% in adulthood.[55] Isthmic spondylolysis occurs as a stress fracture of the pars interarticularis as a result of repetitive stress.[56] Several anatomic and biomechanic factors concentrate the stress of upright posture on the lumbosacral junction making L5 the most common site of spondylolysis. The concepts of malleability and adaptability of the child's spine again are exemplified by the capacity of the pars interarticularis to elongate substantially prior to fracture.[57] It is well accepted that in normal stance approximately 20% of the force is transmitted through the posterior elements. In hyperlordosis, that load increases to 50%.[47] The rigidity of the immobile caudal sacral segment articulating with the

long lever arm of the cephalad spine and trunk concentrates stress in the posterior elements at the level of transition.[58] This concentration of stress at the site of transition to a rigid caudal segment also explains why the posterior elements of L4 may be spondylolytic in the presence of a sacralized L5 vertebra.[57]

The pathogenesis of spondylolisthesis in patients with spondylolysis remains controversial. Anterolisthesis is thought by many to occur through the disk. Degeneration of the L5-S1 disk is a commonly identified etiology in adult degenerative spondylolisthesis. However, Farfan et al.,[59] in 1976, theorized that slippage occurs through the epiphyseal endplates of the vertebral body. He sought to explain the fact that slippage has been described to occur in the growth period and to be rare after skeletal maturity. Evidence to support this theory has increased in recent years. An MRI study found that adolescent athletes with spondylolisthesis demonstrated increased endplate lesions between the growth plate and osseous endplate compared with young athletes with spondylolysis without spondylolisthesis.[60] In this same study, wedging of L5 and rounding of the sacrum also progressed with the degree of slippage. Biomechanical, human, and animal studies have also suggested that the weakest part of the immature functional spinal unit is the growth plate.[61–63] Specifically, Sairyo et al. demonstrated in immature calf spines and later in rat spines that failure occurred at the superior growth plate of the caudal vertebra. They further suggest that slippage occurs between the growth plate and the osseous endplate (Fig. 45-9).[64,65] Slippage through this plane could also

Fig. 45-9 The superior physis of the caudal vertebral body is the weakest structure at the lumbosacral junction in the immature calf spine. Spondylolisthesis occurs throughout the physis in response to applied load.

result in rounding of the S1 vertebral body. This rounding of the caudal vertebra was shown to play a central role in progression to spondyloptosis in the series reported by Yue et al.[66] Rounding of S1 was the only anatomic finding noted in all 27 cases of spondyloptosis (Fig. 45-10). A word of caution, however, is warranted before extrapolation of these biomechanical and animal studies to human pathogenesis of spondylolisthesis with spondylolysis. The rat has more clearly developed vertebral physes than do larger primates, including humans. This is in distinction to the ring apophysis that forms in humans and larger primates.[65] The etiology of spondylolisthesis thus remains elusive and further human study is warranted. Of course, it is difficult to study the immature human spine given the paucity of cadaveric specimens and we will likely rely on extrapolation from larger primates in the future.

PROGRESSION OF SPONDYLOLISTHESIS

Treatment decisions in spondylolisthesis are predicated on the ability to predict progression. Progression of spondylolysis and spondylolisthesis, defined as an increase of 20% or more olisthesis, occurs in less than 3% of skeletally immature adolescents and children.[67,68] In those children with spondylolisthesis and an intact pars interarticularis, such as congenital dysplastic posterior elements, progression of deformity and neurologic injury are more common.[69] Forward slippage of one vertebral body on another is a form of spinal instability. This instability under physiologic load occurs when there is a loss of, or attenuation of, the normal restraints to anterolisthesis. In the normal spine the deforming force is body weight. The force of anterolisthesis is countered posteriorly by the posterior osseoligamentous structures and anteriorly by the anterior buttress created by the intact anterior lip of the sacrum. This buttress becomes ineffectual when the sacrum is substantially rounded, when the L5 body is wedged or kyphosed over the sacrum, and when there is more than 50% anterolisthesis.[70] The presence of these features increases the instability and thus increases the likelihood that the lumbosacral spine will be unable to resist physiologic forces resulting in progression of deformity.

Lumbosacral kyphosis, which may occur in spondylolisthesis, displaces the center of gravity anteriorly relative to the level of the slip. The remainder of the spine may then undergo compensatory changes in an attempt to maintain sagittal balance. Commonly expected changes are increased lumbar lordosis and decreased thoracic kyphosis.[71] Seitsalo and colleagues[72] hypothesize that the loss of thoracic kyphosis in high-grade spondylolisthesis may be associated with the development of coronal plane abnormalities in these patients. They suggest that this may explain the occurrence of idiopathic-appearing scoliosis in the spondylolisthesis population.
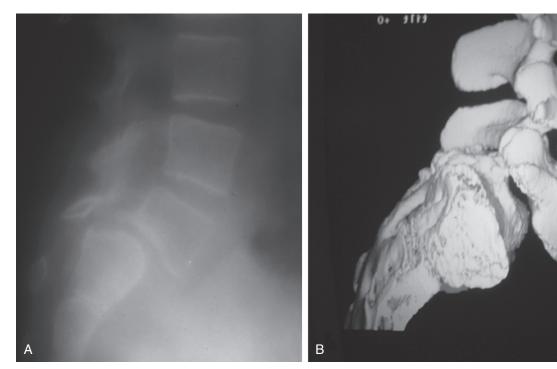
Fig. 45-10 A characteristic finding in spondylolisthesis is rounding of the anterosuperior body of S1 *(A)*. Three-dimensional CT scan demonstrates the rounding of the anterior body of the sacrum *(B)*.

CONCLUSION

A thorough understanding of the biomechanical principles affecting the pediatric spine is crucial for the management of injuries and disorders of the child's spine. The unique anatomic characteristics of the pediatric spine help us to understand the behavior of the spine in response to load. Immature musculoskeletal tissues have significant capability to remodel and adapt to applied loads such as trauma or growth; however, the encased neural structures lack this ability. The elasticity or malleability of the spinal column, the presence of physes, the orientation of the facet joints, and the disproportionate size of the head distinguish the pediatric from the adult spine. These features explain the propensity for upper cervical spine injuries in children younger than 8 years and the increase in osseous lesions in older children and adolescents. These anatomic characteristics help to explain the phenomenon of SCIWORA in the young child and guide the management of trauma and instability in the pediatric spine.

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Imaging Modalities of the Traumatically Injured Pediatric Spine

RADIOGRAPHY

Radiography continues to be the mainstay of any investigation of the pediatric spine, despite rapid advances in other areas of diagnostic imaging. Radiographs provide important information about alignment of vertebral bodies, disk space height, facet joint integrity, trabecular patterning, and cortical bone destruction. Every child with back pain should have at least an anteroposterior (AP) and lateral radiograph of the spine in the area of maximum symptoms. Most traumatic spinal injuries that occur in children younger than 8 years, as many as 90%, are located at C3 or higher. For this reason, thorough evaluation of the cervical spine including open-mouth odontoid, AP, and lateral views are also essential in acute management of every pediatric trauma patient. C-spine assessment is especially important in an obtunded patient or in association with head or facial trauma because of the strong association with significant cervical injury. If the lower cervical vertebrae are not seen on standard lateral views, a swimmer's view with gentle downward arm traction may be obtained. It is important to remember that during transport and assessment, positioning on a backboard with an occipital cutout is mandatory. Children have a relatively large head circumference in relation to the size of their body, so immobilization on a flat backboard can lead to excess cervical flexion and potential spinal compromise.

Films for suspected instability should also include standing flexion and extension lateral views. Flexion-extension views should always be performed with voluntary movement of an awake and alert patient and should not be ordered in severe trauma until the static AP and lateral views have been evaluated. Oblique views can be added if a suspected pars defect is not seen on the lateral view or for closer examination of the facet joints, pedicles, or lateral masses. In addition, any obtunded patient, a patient with a significant mechanism (motor vehicle accident or fall of more than 10 feet), or significant associated injury (major long bone fracture, cervical or head/facial injury) should have thoracolumbar spine radiographs. This evaluation should be undertaken with the knowledge that young children can have significant spinal cord injury, presenting with partial or complete paralysis, even with normal-appearing radiographs. This has been described as the so-called spinal cord injury without radiographic abnormality (SCIWORA) phenomenon.[1]

Radiation exposure, especially to the developing breasts and thyroid, is minimized when possible by use of postero-anterior (PA) projections, fast x-ray tube filtration, beam collimation, and rare earth screens. However, AP radiographs are often preferred in the trauma setting because they generally provide superior bony detail of the vertebrae to that seen on the PA view. If possible, all young women of childbearing age should be asked and if necessary tested for pregnancy before radiographs are performed. Radiation shields to protect reproductive organs should be used whenever possible.

Evaluation of pediatric cervical spine radiographs requires understanding of the common normal variations that occur during spinal development and knowledge of important measurement parameters. The cervical spine approaches adult size by age 8 years, and the vertebral bodies gradually lose their oval or wedge shape and become more rectangular. Thus, children in the first decade are unique; whereas radiographs of older children appear similar to the adult population. Two important parameters to evaluate are the atlas-dens interval (ADI) and the space available for the cord (SAC). ADI represents the distance from the posterior cortex of the anterior arch of the atlas to the anterior cortex of the dens process (Fig. 46-1). In flexion, an ADI of more than 3 mm on lateral radiographs indicates instability in an adult, but in a child up to 5 mm is normal because of ligamentous laxity and unossified cartilage in the dens. In extension, 20% of normal children have riding up of the anterior arch of C1 onto the odontoid process, whereas in adults this is an abnormal finding.[2,3] According to Steel's rule of thirds, space within the spinal canal at the C1 level is one third occupied by the odontoid, one third occupied by the spinal cord, and
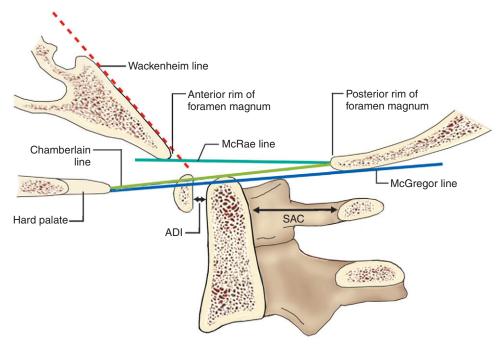
Fig. 46-1 The atlanto-dens interval (ADI) and space available for the cord (SAC) are used for evaluation of atlanto-axial instability. The Wackenheim clivus-canal line is a method of evaluating for atlantooccipital injury. The McRae and McGregor lines are used to evaluate for basilar impression. (From Warren WC, Hedequist DJ: Cervical spine injuries in children. In Beaty JH, Kasser JR: Rockwood and Wilkins Fractures in Children, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2006, pp 775–814. Reprinted from Copley LA, Dormans JP: Cervical spine disorders in infants and children. J Am Acad Orthop Surg 6:204–214, 1998 with permission.)

one third is unoccupied. SAC represents the space from the posterior cortex of the odontoid to the anterior cortex of the C1 posterior arch, which contains both the spinal cord and unoccupied canal space (see Fig. 46-1). In young children, the SAC should be equal to or greater than the width of the odontoid and should be a minimum of 13 mm.

The orientation of facet joint surfaces changes with growth, becoming more vertical. Upper facets (C2 to C4) change from 30 to 70 degrees and lower facets from 55 to 70 degrees.[4] The relatively transverse orientation of the facet joints together with the relative laxity of ligamentous and cartilaginous structures in the pediatric spine results in a physiologic increase in cervical mobility. In 20% of normal children younger than age 8, this increased mobility can cause the radiographic appearance of anterior cervical vertebral displacement when the neck is in flexion, known as pseudosubluxation. In this young age group, C2 can move up to 3 mm or more forward on C3 during neck flexion, whereas in a skeletally mature adult no forward movement of C2 on C3 should be present. Pseudosubluxation is most often seen between C2 and C3 but is also common between C3 and C4. The posterior cervical line of Swischuk, also known as the spinolaminar line, is useful in differentiating pseudosubluxation from pathologic subluxation with instability (Figs. 46-2 and 46-3). This line is drawn from the anterior cortex of the posterior arch of C1 to the anterior cortex

of the posterior arch of C3 on a flexion lateral x-ray.[5] In extension and neutral positions, the anterior cortex of the posterior arch of C2 lies posterior to the posterior cervical line. On lateral flexion x-rays, the appearance of anterior displacement of the C2 vertebral body can be attributed to pseudosubluxation when Swischuk's line passes through, touches, or is less than 1.5 mm anterior to the anterior cortex of the posterior arch of C2. A distance of 1.5 to 2 mm is suspicious for dislocation, and with more than 2 mm a true dislocation is assumed. The most common cause for pathologic anterior dislocation of C2 is a Hangman's fracture (bilateral pedicle or arch fractures) of C2, which should be suspected when a true abnormality is identified.

The cervical vertebrae of an infant ossify from numerous ossification centers that are connected by various synchondroses. The cartilaginous synchondroses have a radiolucent appearance that can be confused with fractures. The most prominent examples of this include the neurocentral synchondroses throughout the spine and especially at the atlas, the dentocentral synchondrosis at the base of the dens process, and the apical epiphysis at the tip of the dens process (Fig. 46-4). Neurocentral synchondroses represent the site of fusion between the ossifying centrum and the neural arches. Neurocentral ossification begins in the lumbar spine and progresses cranially to reach the cervical spine by age 2. The clefts may remain radiographically apparent through the
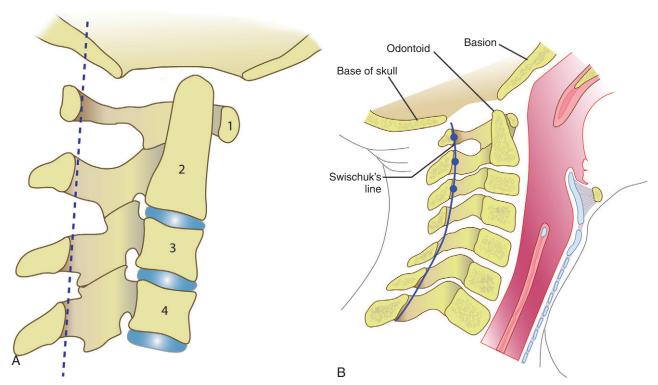
Fig. 46-2 The posterior cervical line (Swischuk's line) differentiates physiologic pseudosubluxation from pathologic instability when translation of C2 on the C3 vertebral body is seen on the flexion-lateral x-ray of the cervical spine. Swischuk's line is drawn between the anterior cortex of the posterior arches of C1 and C3, and stability is determined by its relationship to the C2 posterior arch. (From Madigan RR: Evaluation of the pediatric spine patient. In Fardon DF, Garfin SR (eds): Orthopaedic Knowledge Update Spine 2, Rosemont, IL, American Academy of Orthopaedic Surgeons, 2002, pp 109–121. Warren WC, Hedequist DJ: Cervical spine injuries in children. In Beaty JH, Kasser JR (eds): Rockwood and Wilkins Fractures in Children, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2006, pp 775–814.)

third to sixth year of life. The neurocentral synchondrosis of the atlas persists for longer, fusing at approximately age 8.

The site of fusion between neural arch ossification centers can be seen as a midline posterior sagittal cleft on AP radiographs. The neural arches fuse in the atlas by age 4, in the axis by age 3 to 6, and at other levels by age 2 to 3. Failure of the neural arches to fuse at the posterior synchondrosis can result in a persistent vertical radiolucent line indicating a bifid posterior arch. In addition, failure of posterior arch formation can result in spina bifida occulta. The neurocentral synchondroses of the axis ossify between 4 to 7 years of age. The subdental synchondrosis is well below the most common site of dens fracture, which is at the level of the articular processes of the axis. The subdental synchondrosis usually has fused by 6 to 7 years of age but may persist as a sclerotic line until age 11.[2] The tip of the odontoid ossifies between years 2 to 6 and fuses with the main mass of the odontoid by age 11 to 12.

The annular ossification centers of the superior and inferior vertebral body margins, known as the ring apophyses, appear by approximately age 7. The superior and inferior ring apophyses on any given vertebra may not appear simultaneously and they generally do not fuse with the vertebral body until about age 18. Occasionally, lumbar level ring apophyses may persist into the third decade. Ring apophyses are most easily seen on lateral x-ray, where they appear as thin sclerotic lines along the end plates with associated slight vertebral body beaking or notching at the apophyseal edges. In addition, spinous and transverse processes may have secondary ossification centers that appear at approximately the time of puberty and do not fuse to the main mass of the neural arches until about age 25.

Before fusion begins, the epiphyseal plates can be distinguished by the distinctive smooth, incongruent appearance of their margins and their anatomic location. Fractures tend to be irregular, sharp, congruent, and situated in different locations than synchondroses. The presence or absence of focal tenderness on clinical examination can also help make the distinction. Other normal anatomic variants include ventral and dorsal clefts seen within the oval-shaped centrum, which are vascular channels. The anterior notch is formed by a large sinusoidal blood space within the vertebral
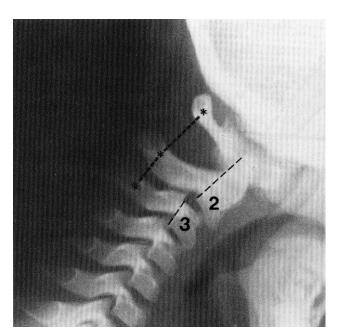
Fig. 46-3 Pseudosubluxation of C2 on C3. Measurement of vertebral bodies in children under age 8 *(thin dashed line)* are unreliable. The relationship of the posterior elements is more consistent *(thick dotted line)* forms and is used in the form of Swischuk's line to assess for true instability. (From Swischuk LE: Anterior displacement of C2 in children: physiologic or pathologic? Radiology 122:759–763, 1977.)

ossification center, whereas the posterior one is formed by a perforated indentation on the posterior wall of the vertebral body functioning as a conduit for basivertebral veins and nutrient arteries.[6] Cartilaginous plates are routinely observed on magnetic resonance imaging (MRI) and occasionally on radiographs at the site of sclerotomal fusion containing the intersegmental vessels. The ventral indentation gradually disappears with age, but the dorsal indentation may remain radiographically and functionally significant into adulthood. In addition, mach bands are an optical phenomenon that can be mistaken for a fracture. These dark and light lines appear at the borders of structures with different radiodensity and are known to occur across the base of the dens.

Increased prevertebral space between the anterior vertebral body of C3 and the laryngeal air column is a helpful indicator of cervical spine injury in adults. However, prevertebral (retropharyngeal) swelling is unreliable in infants and children. Although the distance is increased with bleeding, infection, or edema, the normal space seen as a soft tissue shadow on x-ray varies significantly with neck flexion, extension, and respiration (especially expiration as seen in the crying child). The normal retropharyngeal space in adults does not exceed 3 mm anterior to C3 but is as great as 7 mm in children. The retrotracheal space anterior to the body of C6 is generally less reliable than that at C3 and is normally 8 to 10 mm in adults but is as wide as 14 mm in children.

Despite the increased variability, this is still an important finding because abnormal values beyond this range are highly suggestive of injury.[7]

Abnormalities of the sagittal cervical contour such as angulation at a single interspace, absence of normal cervical lordosis, and absence of cervical kyphosis from C2 to C7 on maximal flexion can also be normal findings in 14% to 16% of children. However, it is important to remember that subtle signs, such as reversal of normal cervical, thoracic, or lumbar sagittal curves and evidence of soft tissue swelling, may be the only indication of significant ligamentous disruption. Spontaneous reduction, protective myospasm, and immobilization devices can all mask significant soft tissue injury on plain x-ray and so these findings remain an essential part of the evaluation.

The atlanto-occipital interval is the most challenging area to assess in a child's spine because of difficulty in obtaining quality x-rays and a lack of discrete reproducible landmarks. In addition, cervicobasilar abnormalities can be due to multiple etiologies that must be recognized, including occipital hypoplasia, trauma, tumor, infection, abnormal cranial ossification, and generalized bone disease. The anterior cortical margin of the foramen magnum is known as the basion, and the posterior cortical margin is termed the opisthion. The relationship of these structures to the atlas is useful for identifying atlanto-occipital injuries. The middle half of the odontoid lies directly beneath and is an average of 5 mm inferior to the basion. In infants and young children, this distance may be as much as 1 cm. Wholey et al.[8] state that a variation greater than 1 to 2 cm in the relationship of the odontoid to the basion requires further evaluation. Whereas, Bulas et al.[9] indicate that the distance on lateral x-ray between the basion and the tip of the dens process should be less than 12 mm. The distance between the occipital condyles and the facet joints of C1 is normally less than 5 mm, with greater distances suggesting atlanto-occipital joint disruption.[10,11] Widening of the spinous processes between C1 and C2 of more than 10 mm also is indicative of a ligamentous injury and should be evaluated by further imaging studies.[12]

The Powers ratio is another method used to assess the relationship of the skull base relative to the atlas. The ratio is determined by drawing a line from the basion to the anterior cortex of the posterior arch of C1 and dividing this distance by that of a line drawn from the opisthion to the posterior cortex of the anterior arch of C1 (Fig. 46-5). Normal values range between 0.7 and 1 with higher values indicating anterior subluxation of the atlanto-occipital joint and a lower value indicating posterior subluxation. The major difficulty with this measure is that the basion is not always clearly visualized on plain x-ray. In addition, the Powers ratio may be insensitive to posterior occipitoatlantal dislocation and is not valid if there is a fracture of the atlas or congenital anomaly of the foramen magnum.[13,14] The most easily identified alternative measure to assess for atlanto-occipital joint
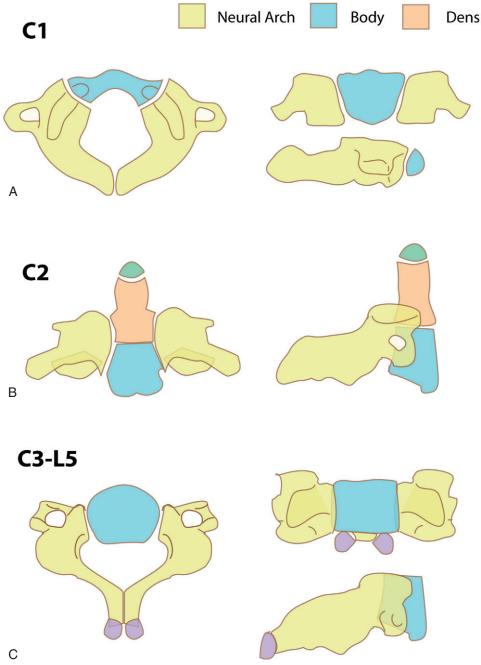
Fig. 46-4 *A,* Ossification centers of C1. *B,* Ossification centers of C2. *C,* Ossification centers typical of C3 to L5. (From Sullivan JA: Fractures of the spine in children. In Green NE Swiontkowski MF (eds): Skeletal Trauma in Children, 3rd ed. Philadelphia, Saunders, 2003, pp 472–515.)

disruption is the Wackenheim line, which is drawn along the posterior aspect of the clivus (see Fig. 46-1). If the line does not intersect the tip of the odontoid tangentially and if this line is displaced anteriorly or posteriorly, disruption or increased laxity about the atlanto-occipital joint should be suspected.

The overall alignment of the lower cervical spine can be evaluated by examining four continuous lines including the line adjoining the spinous processes, the spinolaminar line, and the lines adjoining the posterior and anterior vertebral bodies. These lines should all be smooth and continuous with no evidence of vertebral translation (Fig. 46-6). The interspinous distance at each level should be evaluated and should be no more than 1.5 times the distance at adjacent levels; if this ratio is greater, an injury should be suspected. There are calculated norms for the interspinous distances in
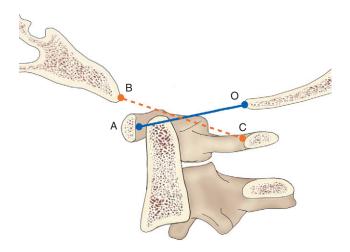
Fig. 46-5 The Powers ratio is determined by drawing lines from the basion (B) to the posterior arch of the atlas (C) and from the opisthion (O) to the anterior arch of the atlas (A). The length of line BC is divided by the length of line OA (BC/OA), producing the Powers ratio. (From Warren WC, Hedequist DJ: Cervical spine injuries in children. In Beaty JH, Kasser JR: Rockwood and Wilkins Fractures in Children, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2006, pp 775–814. Reprinted from Lebwohl NH, Eismont FJ: Cervical spine injuries in children. In Weinstein SL (ed): The Pediatric Spine: Principles and Practice. New York, Raven, 1994, pp. 725–742; with permission).


Fig. 46-6 Normal relationships in the lateral cervical spine: 1, spinous processes; 2, spinolaminar line; 3, posterior vertebral body line; 4, anterior vertebral body line. (From Warren WC, Hedequist DJ: Cervical spine injuries in children. In Beaty JH, Kasser JR: Rockwood and Wilkins Fractures in Children, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2006, pp 775–814. Reprinted from Copley LA, Dormans JP: Cervical spine disorders in infants and children. J Am Acad Orthop Surg 1998; 6:201–214 with permission.)

children, and any value greater than two standard deviations above normal is indicative of a ligamentous injury[15] (Table 46-1).

COMPUTED TOMOGRAPHY

Ideally, computed tomography (CT) is used to further define pathology noted on plain radiographs or bone scan and is not a primary screening modality unless quality radiographs cannot be obtained. The diagnostic characteristics, exact location, and real or potential instability of fractures can be assessed. CT is superior to MRI in demonstrating osseous injury and bony detail. CT of a fractured vertebra will provide the information needed to classify the fracture as stable or unstable and determine whether operative treatment is needed. It is also useful for determining the adequacy of spine fusion or healing of pars defects. In addition, it is especially useful for identifying lesions of the facets and posterior elements, defining burst fractures of the vertebral bodies, and assessing the spinal canal.

CT scanner technologic advances have allowed for improved image resolution, reduction in acquisition time, and software for three-dimensional (3-D) reconstruction. CT images obtained in the axial plane are the standard, but with the development of spiral CT allowing the collection of volumetric data, reformat-

ted images in the coronal and sagittal plane are common and add valuable information. Most recently, software has become widely available that allows a graphic computer to produce 3-D reconstructed images from the data collected during axial imaging. 3-D CT greatly simplifies investigation of complex problems in the craniocervical junction of children. 3-D CT is also a valuable examination for spinal injuries when congenital abnormalities are present or with other complex injury patterns.

CT scan is an essential tool for evaluation of atlantoaxial rotatory subluxation. When atlantoaxial rotatory displacement is noted on plain radiographs (open mouth or odontoid view), it is very difficult to determine if the subluxation is fixed on plain x-ray. Fixed subluxation is confirmed by

TABLE **46-1** Normal Ossification Centers and Anomalies Frequently Confused with Injury

Avulsion fracture

    Apical ossification center of the odontoid

    Secondary ossification centers at the tips of the transverse and spinous processes

Fracture

    Persistence of the synchondrosis at the base of the odontoid

    Apparent anterior wedging of a young child's vertebral body

    Normal posterior angulation of the odontoid seen in 4% of normal children

Instability

    Pseudosubluxation of C2-C3

    Incomplete ossification, especially of the odontoid process, with apparent superior subluxation of the anterior arch of C1

    Absence of the ossification center of the anterior arch of C1 in the first year of life may suggest posterior displacement of C1 on the odontoid

    Increase in the atlanto-dens interval of up to 4.5 mm

Miscellaneous

    Physiologic variations in the width of the prevertebral soft tissue due to crying misinterpreted as swelling due to edema or hemorrhage

    Overlying structures such as ears, braided hair, teeth, or hyoid bone. Plastic rivets used in modern emergency cervical immobilization collars can simulate fracture line

    Horizontally placed facets in the younger child, creating the illusion of a pillar

fracture

    Congenital anomalies such as os odontoideum, spina bifida, and congenital fusion or hemivertebrae

From Warren WC, Hedequist DJ: Cervical spine injuries in children. In Beaty JH, Kasser JR: Rockwood and Wilkins Fractures in Children, 6th ed. Philadelphia, Lippincott Williams & Wilkins, 2006, pp 775–814.

dynamic CT scan with axial cuts at the C1-C2 area. Fixed subluxation is diagnosed when both the initial scan and the scan repeated with the head maximally turned in the opposite direction show continued rotatory subluxation.

MAGNETIC RESONANCE IMAGING

MRI is the diagnostic modality of choice for evaluating soft-tissue intraspinal and paraspinal abnormalities in infants, children, and adolescents. MRI is superior to CT in demonstrating spinal cord pathology and intervertebral disk herniation. In many severe injuries, especially SCIWORA, the plain radiographs are normal. Even if a fracture is present on x-ray, a soft tissue or cord injury at a different level may be present. For this reason, indications for MRI include cervical level spinal cord injury, any incomplete spinal cord injury, and suspected disk or ligament injury. MRI is also increasingly valuable in allowing mobilization of the cervical spine in patients unable or unwilling to cooperate with flexion-extension radiographs.[16]

Accurate assessment of spinal cord parenchymal lesions by MRI improves the accuracy of prognosis following trauma and can demonstrate cord compression from bony fragments, vertebral canal malalignment, and disk extrusion. Findings on MRI correlate well with outcome of spinal cord injury. Spinal cord transaction is presently irreversible and

cord hemorrhage is nearly so, while patients with edema have potential for recovery of neurologic function. Contusion is often associated with an incomplete lesion, whereas compression patterns are likely to be associated with either complete paralysis or an incomplete lesion with little likelihood of improvement. In addition, MR angiography has replaced standard arteriograms in children with upper cervical spine injury and suspicion of associated vascular damage.[1]

Knowledge of normal developmental anatomy is vital to proper interpretation of MRI in infants and small children. The conus can normally extend to the body of the third lumbar vertebra in infants 3 months of age and younger. After 3 months, the conus should lie at the L1-L2 level. In children younger than 3 years, multiple changes alter MRI signal characteristics including ossification of the vertebral cartilaginous endplates, change from red to yellow marrow in the vertebrae, weight-bearing stresses on the spine as the infant begins to sit, stand, and walk, and changing water content of the disks.

In the first 6 months of life, the bone marrow of the vertebral body is ovoid in shape with hypointense signal on both T1- and T-2 weighted images. The cartilaginous endplates are hyperintense on T1-weighted images and hypointense on T2-weighted images. The disks remain hypointense on T1-weighted images and hyperintense on T2-weighted images throughout growth. Between months 6 to 12, the bone marrow of the vertebral body becomes more rectangular and

fatty cells begin to infiltrate. This causes the signal intensity of the vertebral body to become more intense compared with the cartilage and disk on T1-weighted images. The cartilaginous end plates become ossified by this age and demonstrate no signal on T1- or T2-weighted images in areas of ossification. By 2 years of age, each part of the vertebra is more distinct, and normal spinal curvature is established. Continued fatty infiltration of the marrow causes a more marked hyperintense signal on T1-weighted images and a hypointense signal on T2-weighted images of the vertebral body. The endplates are ossified and demonstrate no signal on T1- or T2-weighted images. Signal characteristics of the disk may change as the child continues to age, with approximately 25% of asymptomatic adolescents demonstrating MRI evidence of disk degeneration.

It is difficult to obtain quality MRI images in children without sedation to suppress voluntary motion artifact. Children younger than 12 years usually require heavy sedation or low-dose general anesthesia, both of which increase the morbidity of the procedure. Because of the risk of sedation and the increased cost of testing, MRI should not be used as a screening tool. It should be reserved for patients whose clinical symptoms and signs point to an underlying soft-tissue abnormality or in clinical situations with a high incidence of associated neurologic abnormalities.

RADIONUCLIDE SCAN

Radionuclide scans are useful for evaluation of infection, tumor, stress reaction, and some fractures. Gallium and indium are commonly used to evaluate infection, but technetium (Tc) 99m "bone" scan is most commonly used for evaluation of spine problems. The delayed bone phase is the most useful because it measures the level of active bone turnover. The metabolically active area of growing physes shows modestly increased uptake in growing children. Pathologic areas of increased turnover can be due to tumor, infection, or fracture. Radionuclide studies currently have a limited role in the acute management of the multiply injured child, although Heinrich et al. reported 19 previously unrecognized fractures in 48 patients investigated with bone scan. Six of these injuries (12.5%) caused a change in management including casting of nondisplaced fractures after further radiographic investigation. When occult fracture, stress reactions or fractures, diskitis, or bone neoplasm are suspected, the bone scan should be the next imaging modality selected after the plain radiograph.

Single-photon emission CT (SPECT) is a technique that essentially uses CT technology for evaluation of bone scintigraphy. SPECT improves the diagnostic ability of bone scintigraphy by permitting separation of bony structures that overlap in standard planar images, resulting in precise localization of the anatomic area involved. SPECT is the modality with the highest degree of both sensitivity and specificity in detecting stress lesions/fractures in the pars interarticularis.

Approximately 25% of lesions present in SPECT will be seen on plain radiographs, and planar bone scintigraphy will show only 50%.

ULTRASOUND

Because sound waves do not penetrate bone, ultrasonography is not effective for spinal cord imaging in the older infant and child. However, the unossified midline posterior elements of the vertebral column in the neonate and infant (up to 3 to 6 months of age) provide an acoustic window for visualizing the spinal cord, filum terminale, nerve roots, cauda equine, and distal thecal sac.[17] Spinal ultrasound is indicated in infants with physical findings that suggest an underlying dysraphic lesion and infants with caudal regression or anorectal malformations that carry a risk of cord tethering. Intraoperative ultrasonography has increased acceptance in the neurosurgical community to accurately measure the extent of cord tumors and cystic lesions.

MYELOGRAPHY

Myelography has been almost completely replaced by MRI in the evaluation of the pediatric spine, except in specific circumstances. When MRI is not available or cannot be done, myelography with water-soluble contrast material may be helpful in assessing extrinsic cord compression, particularly in patients with incomplete lesions. In addition, the CT myelogram is the test of choice to evaluate for cord compression when spinal hardware is already present. Other indications for myelography include: identification of the exact anatomy of congenital anomalies, such as diastematomyelia, meningoceles, or other defects before corrective surgery; documentation of drop metastases from tumors such as medulloblastomas, ependymomas, and pineal region tumors when contrast-enhanced MRI study is negative; confirmation of traumatic avulsion of the nerve sheath and posttraumatic pseudomeningocele.

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C H A P T E R

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Pediatric Spinal Cord Injury and Spinal Cord Injury Without Radiographic Abnormality (SCIWORA)

INTRODUCTION

Each year, approximately 10,000 people in the United States present with a spinal cord injury and approximately 150,000 to 160,000 present with a spinal column fracture.[1,2] Pediatric spine injuries are relatively rare: pediatric patients account for only between 1% and 11% of the large number of patients presenting with a spine injury each year.[3–19] Anatomic and biomechanical differences between children and adults, such as the child's smaller size and the fact that children are growing, lead to different patterns of injury than is seen in adults. For instance, one type of injury almost unique to children (although it can also on occasion be seen in adults) is spinal cord injury without radiographic abnormality (SCIWORA), which was first described by Pang and Wilberger in 1982.[20,21] It is a traumatic process that occurs in children who present with findings of neurologic injury but without evidence of fracture or dislocation on conventional radiographs and excludes injuries from penetrating trauma, electrical shock, obstetric complications, and injuries associated with congenital spinal anomalies.[20–23] SCIWORA by definition is more difficult to detect, and therefore, may be more difficult to treat. Other injuries, such as atlanto-occipital dissociation, upper cervical spine injuries, and vertebral end-plate fractures, also occur more often in children than in adults.[3–18] The unique nature of these injuries, combined with their relatively rare occurrence, make the detection

and treatment of spine injuries in children more challenging and may at times result in incorrect diagnosis and treatment.

EPIDEMIOLOGY

The annual incidence of acute spinal cord injuries for patients admitted to acute care hospitals in the United States has been estimated to range between 3.2 to 5.3 per 100,000 persons, and currently approximately 250,000 people in the United States are living with some degree of spinal cord injury.[1,19] An acute spinal cord injury estimated to be present in approximately 2.6% of all major trauma victims; of these between 43% to 46% result in complete loss of sensory and motor function below the level of the injury.[24,25] Approximately 55% of these injuries occur in the cervical spine, 30% in the thoracic spine, and 15% in the lumbar spine. Of the patients with injuries to the cervical spine, approximately 40% present with a complete spinal cord injury, 40% with an incomplete spinal cord injury, and the remaining 20% present with either no cord injury or only a root lesion.[24,26] The prevalence of spinal column injury is bimodal with peaks occurring in people between 15 and 24 years of age and in people 50 years and older with an overall mean age of 33 years.[1,19] Pediatric spinal injuries, depending on the study, account for only 1% to 11% of all spinal injuries; however, it is estimated that approximately 5% of all spinal injuries occur in children aged birth to 16 years of age resulting in approximately 500 to 1000 reported spinal cord injuries in the pediatric population.[3–19]

Epidemiologic studies have found that the type, location, cause, and severity of spinal injuries in the pediatric population vary with age of the patient. Osenbach and Menezes[6] found in their review of 179 children aged birth to 16 years of age that pediatric spinal cord and/or vertebral column injuries accounted for 9% of all spinal trauma during an 18-year study period. Sixty-two of the 179 children were between birth and 8 years of age and 117 were between 9 and 16 years of age. The younger group of children had a higher incidence of cervical spine injury than the group of older children (79% vs. 54%, respectively), and the younger group also sustained injuries to the upper cervical spine twice as

often as the older group. Lower cervical and thoracic injuries were equally frequent in both groups, but thoracolumbar injuries were more common in the older group. Nondisplaced fractures were more common in older children, but subluxation without fracture and SCIWORA were more frequent in the younger patients. The incidence of neurologic deficit was higher in the younger group than in the older group (62% vs. 47%, respectively). Children with complete or severe incomplete myelopathy uniformly remained with severe neurologic deficits, whereas children with mild to moderate injuries recovered normal or nearly normal neurologic function.[6]

Hadley et al.[7] found in their review of 122 cases of pediatric spinal cord and vertebral column injury an increasing incidence of spinal injury with increasing age. In their report, 18 patients were birth to 9 years of age, 38 patients were 10 to 14 years of age, and 66 patients were 15 to 16 years of age. The most common causes of injury in the birth to 9 years of age group were pedestrian-automobile accidents and falls, whereas the most common causes of injury in the older group were motor vehicle crashes and sports-related trauma. Similar etiology of pediatric spinal injury was found by Ruge et al.[9] who found in their review of 72 children aged 12 year or younger that falls were the most common causative factor of spinal injury accounting for 38% of cases, followed by automobile-related injuries, which accounted for 20% of cases. Hadley et al.[7] found in their review a higher incidence of neurologic injury and SCIWORA in the youngest age group. The cervical spine accounted for 72% of injuries in the birth to 9 years of age group and 50% were centered between the occiput and C2. The older children were injured at levels observed in the adult population.[7] Apple et al.[18] in their analysis of 1770 traumatic spinal cord injury patients found that the most common level of spinal injury was C2 in preteens, C4 in teens, and C4-C5 in adults.

Hamilton and Myles[5] found that pediatric spine injuries accounted for 5.4% of all spinal trauma and that younger patients were found to have a higher incidence of neurologic injury and SCIWORA. As did Osenbach and Menezes,[6] the authors found that children with complete cord injuries showed little improvement, whereas patients with incomplete injuries fared better with 74% showing significant improvement and 59% showing complete recovery.[5] More recently, Carreon et al.[16] found in their review of 137 children with spine injuries that the incidence of spine injury increased with age: 36 children were younger than 10 years, 49 were 10 to 14 years of age, and 52 were 15 to 17 years of age. In their analysis, motor vehicle accidents were the most common overall cause of spine injury. Nineteen percent of the patients had a spinal cord injury, and spinal cord injury was more common in the youngest age group.[16] The higher incidence of spinal cord injury, and perhaps more importantly, greater severity of spinal cord injury in younger patients, has recently been confirmed by an analysis of the Shriners Hospitals for Children spinal cord injury database and the National Spinal Cord Injury Statistical Center database from 1973 to 2002, in which DeVivo and Vogel[17] found that spinal cord injury was much more likely to be neurologically complete in younger children (69% in children birth to 5 years of age vs. 51% in children 16 years of age or older).

With respect to SCIWORA specifically, the incidence among children with traumatic spinal cord injury varies widely. Earlier reports of the incidence have ranged between 1.3% and 67.1%,[15,21,27,28] whereas a recent comprehensive review of the topic by Pang[23] reported a mean incidence of SCIWORA in children with traumatic myelopathy of 34.8% (range, 4.8%–67.1%) in 15 studies. Both the incidence and the severity of SCIWORA are inversely related to the age of the child primarily because of the developmental and biomechanical factors described later.[6,7,28–30] The most common mechanisms of injury leading to SCIWORA include motor vehicle accidents, falls from a height, sports-related injury, and child abuse.[23,28]

Several conclusions can be drawn from epidemiologic studies of pediatric spinal injuries. The incidence of pediatric spinal injury increases with age and appears to be greatest in children 15 to 18 years of age. In children younger than 8 years, most series have found that spinal injuries tend to be centered in the upper cervical spine and are more often ligamentous. Higher prevalence of injuries caused by falls, pedestrian automobile injuries, child abuse, and birth injuries have been noted in this age group than in older children. Spinal cord injury, including SCIWORA, is more common in younger rather than older children, and incomplete spinal cord injuries are more likely to improve in younger children. In contrast, in children older than 8 to 9 years, spinal injuries begin to resemble the epidemiology and pattern of injury similar to that of adult patients.[3–18]

BIOMECHANICS AND PATHOPHYSIOLOGY

Age-related anatomic differences between children and adults play an important role in explaining the different patterns of spinal injuries seen in pediatric as opposed to adult patients. The most obvious difference is the relative cephalocervical disproportion; In infants, for example, the weight of the head may account for as much as 25% of the total body weight, whereas in adults, the head accounts for only approximately 10% of the total body weight. In addition to the proportionally large and heavy head, the cervical and nuchal musculature is not well developed in young children. The combination of a large head and weak muscle support predisposes the pediatric spine to greater motion when subjected to flexion and extension forces.[14,23,31] In addition to this most obvious difference between children and adults, other factors have been described in multiple prior studies, and have recently

been summarized concisely by Pang.[23] These factors include (1) ligamentous and joint capsule laxity; (2) relatively high intervertebral disk and annular water content, allowing for lengthening of the spinal column; (3) relatively shallow and horizontally oriented facet joints, which allow for increased translation, flexion, and extension; (4) anterior wedging of vertebral bodies; and (5) absence of the uncinate processes.[23] The net effect of these anatomic differences—which begin to decrease in importance as the pediatric spine starts to resemble the adult spine at approximately 8 to 9 years of age—is that children have less skeletal resistance to flexion, extension, and rotational forces with a greater degree of resistance being shifted to the ligaments. This helps to explain why children younger than 8 years are more likely to have spinal injuries, which are ligamentous in nature, and are more likely to have SCIWORA.[14,23]

CLINICAL PRESENTATION

The scope of pediatric spinal injury can be categorized into four major patterns: vertebral column fracture, fracture with subluxation/dislocation, subluxation/dislocation alone, and SCIWORA. Spinal cord injury, as evidenced by a neurologic deficit, may be present regardless of the specific injury pattern (and is by definition present in SCIWORA). Hadley et al.[7] found that approximately 31% of their patients (38 of 122) presented with an incomplete spinal cord injury and approximately 16% of their patients (20 of 122) presented with a complete spinal cord injury. Of the 58 patients, that presented with a neurologic deficit, 33% had SCIWORA. The younger patients (aged from birth to 9 years of age) were more likely to sustain neurologic injury including SCIWORA.[7] Carreon et al.[16] reported similar findings: the authors noted a higher incidence of spinal cord injury among children aged from birth to 9 years of age compared with older children, and approximately 25% of patients with a spinal cord injury had SCIWORA. The findings have been confirmed by a recent comprehensive review of the topic by Pang[23] who reported a mean incidence of SCIWORA in children with traumatic myelopathy of 34.8% (range, 4.8 to 67.1%). The distribution of the underlying injury patterns is variable. Hadley et al.,[7] for example, found in their review of 122 children aged from birth to 16 years of age with spinal injuries that fractures alone accounted for 41% of cases, fractures with subluxation accounted for 33%, subluxation alone accounted for 10%, and SCIWORA accounted for 16%. Carreon et al.,[16] on the other hand, found in their review of 137 pediatric patients with spine injuries that fractures alone accounted for 78% of cases, fractures with subluxation or dislocation accounted for 12%, subluxation or dislocation alone accounted for 6% of cases, and SCIWORA accounted for 3% of cases.

Pediatric spine fractures and subluxations and dislocations—all of which are covered in more detail in other chapters—may involve the upper cervical spine, subaxial cervical spine, thoracic spine, and the lumbar spine. The most common fractures (with or without subluxation or dislocation) in the upper cervical spine include fractures of C1, such as a Jefferson fracture, or fractures of C2 such as an odontoid fracture (which most often occurs through the dentocentral synchondrosis) or traumatic spondylolisthesis of C2 on C3 (i.e., Hangman's fracture). Unlike upper cervical spine fractures, which can be seen in all pediatric patients regardless of age, subaxial cervical spine fractures tend to occur in older children whose spines resemble that of adults.[14,16,32] When a subaxial cervical spine fracture occurs in younger children, isolated wedge compression fractures may be seen, whereas in older children, subaxial cervical fractures tend to be more complex with both bony and ligamentous injury resulting in fracture with subluxation or dislocation and associated spinal cord injury.[14] Thoracolumbar fractures can occur in both younger and older pediatric patients, but are more common in patients older than 8 years of age.[14,16,32] The most common thoracolumbar fractures include: compression fractures, burst fractures, Chance fractures, and vertebral apophyseal fractures.[14,16,32,33] Chance fractures—which are sometimes referred to as lap-belt fractures—are flexion-distraction injuries, which involve all three columns of the spine and are therefore usually unstable.[14,16,32,33] They can be treated with bracing, casting, and surgery depending on the nature and magnitude of injury (Fig. 47-1, *A* and *B* and Fig. 47-2 *A* to *E*). Vertebral apophyseal fractures, which are sometimes referred to as limbus fractures, classically present clinically like a herniated nucleus pulposus and are typically seen in adolescents. Pediatric subluxation or dislocation injuries include atlantooccipital dislocation (Fig. 47-3, *A* and *B*), transverse atlantoaxial ligament ruptures, atlantoaxial rotatory displacement, and other injuries (Fig. 47-4, *A* to *C*). Atlantooccipital dislocation is a rare and usually fatal injury, which in children may be associated with air bag deployment. Patients are usually polytrauma victims and present with a range of neurologic symptoms from completely asymptomatic to cranial nerve dysfunction to complete loss of function below the brainstem.[10,11,14,31,33] Transverse atlantoaxial ligament ruptures may occur after severe or mild trauma as a result of disruption of the transverse ligament and/or fracture of the odontoid. Atlantoaxial rotatory displacement is usually insidious in onset or may be associated with trivial trauma and may be due to inflammation (Grisel's syndrome), trauma, neoplasia, and other causes.[11,14,33]

Patients with spinal cord injury—regardless of the underlying injury pattern (i.e., vertebral column fracture, fracture with subluxation/dislocation, subluxation/dislocation alone, and SCIWORA)—may present with a variety of neurologic deficits depending on the severity and mechanism of injury. Pediatric spinal cord lesions may be complete or incomplete.[34] A child who has a complete spinal cord injury will
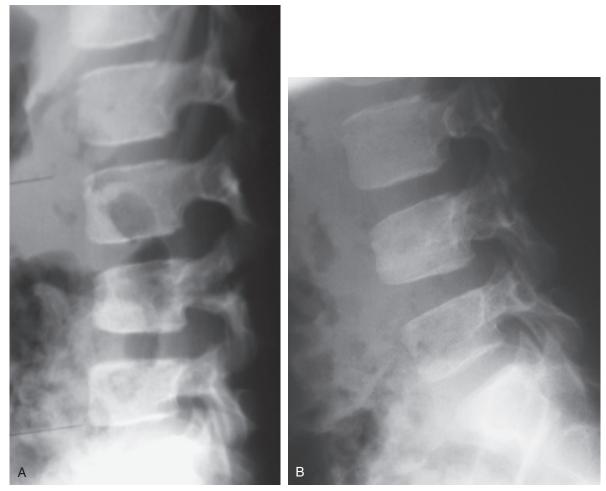
Fig. 47-1 *A,* Lateral radiograph demonstrating a Chance fracture. *B,* The patient was neurologically intact and was treated with a hyperextension cast with good results 2 years following the injury.

have no sensory or motor function distal to the level of the injury, and if the lesion is complete and remains so for 48 hours, return of sensory and/or motor function is very unlikely. A child who has an incomplete spinal cord injury, on the other hand, will have preservation of some sensory and/or motor function distal to the level of the injury, and partial recovery of sensory and/or motor function is likely.[4–7,35] Incomplete lesions may further be classified based on the anatomic area of the cord injured.[34] Central cord syndrome is the most common incomplete cord lesion and typically results from a hyperextension injury with compression of the spinal cord between the ligament flavum posteriorly and the intervertebral disk and posterior aspect of the vertebral body anteriorly. This causes central cord hemorrhage and compression of the adjacent central portions of the corticospinal and spinothalamic white-matter tracts. Patients with central cord syndrome present with variable sensory loss and proportionally greater loss of motor function to upper extremities than lower extremities. More specifically, there may be partial flaccid paralysis of the upper extremities and spastic paralysis of lower extremities, indicative of lower

motor neuron involvement in the upper extremities and upper motor neuron involvement in the lower extremities.[34] Brown-Sequard syndrome results from injury to either the right or left half of the spinal cord: the syndrome may, for example, be caused by unilateral facet fracture and/or dislocation or hemitransection of the cord secondary to a penetrating injury such as a stab wound or gunshot wound. The injury results in unilateral damage to the spinothalamic, corticospinal, and dorsal column tracts. Patients with Brown-Sequard syndrome present with ipsilateral motor paralysis and lack of proprioception, deep pressure, and vibratory sensation and contralateral lack of pain and temperature sensation. The syndrome carries a favorable prognosis with reported substantial recovery of functional walking and motor strength.[34] Anterior cord syndrome is related to vascular insufficiency—typically the anterior spinal artery—and results in damage to the anterior two thirds of the cord with sparing of the posterior columns. The syndrome is usually seen after a flexion injury, fracture-dislocation, or a burst fracture. Patients with anterior cord syndrome present with complete motor paralysis and lack of sharp pain and

temperature sensation because of damage of the corticospinal and spinothalamic tracts, respectively. Because the posterior columns are preserved, patients retain proprioception and the ability to sense vibration and deep pressure. Anterior cord syndrome has a poor prognosis with a minimum chance of return of functional walking.[34] Posterior cord syndrome is a rare syndrome and is characterized by damage of the dorsal columns. This results in loss of proprioception and the ability to sense vibration and deep pressure, and although patients have full motor strength, functional walking is difficult—and sometimes impossible—because of dorsal column function impairment.[34] Although it is useful to classify incomplete spinal cord lesion based on the anatomic area of the cord injured, not infrequently this proves to be difficult as some patients do not have a distinct combination of weakness and/or sensory disturbance to make classification possible (or reliable). In such cases, the lesion is classified as a partial cord syndrome.

Patients with SCIWORA may present with complete or incomplete lesions. In a recent meta-analysis of 392 published cases of SCIWORA, Launay et al.[28] found five different clini-

cal presentations: partial cord syndrome in 55% of patients, complete cord injury in 27%, central cord syndrome in 10%, Brown-Sequard syndrome in 5%, and anterior cord syndrome in 3%. Pang[23] in his review of SCIWORA found that partial cord syndrome was present in 36% of patients, central cord syndrome was present in 29% of patients, complete cord injury was present in 22% of patients, and Brown-Sequard syndrome was present in 13% of patients. There were no cases of anterior cord syndrome in his series.[23]

RADIOGRAPHIC FINDINGS

As with any injury or suspected injury to the spinal column, radiographic imaging begins with conventional anteroposterior and lateral radiographs of the cervical, thoracic, and/or lumbar spine. Thorough evaluation of the radiographs is necessary and should attempt to rule out the presence of subluxation, fracture, dislocation, and soft-tissue injury. Computed tomography (CT) and magnetic resonance imaging (MRI) can be helpful by providing
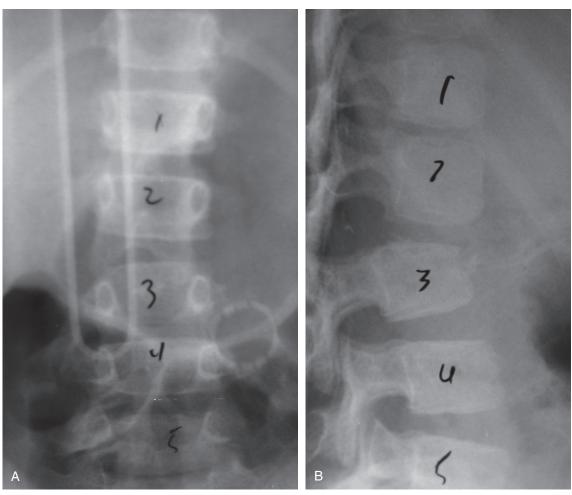

Fig. 47-2 Anterior-posterior *(A)* and lateral *(B)* radiographs demonstrating a Chance fracture in an 11-year-old girl. The patient had a complete spinal cord injury. She was treated with a short-segment fusion
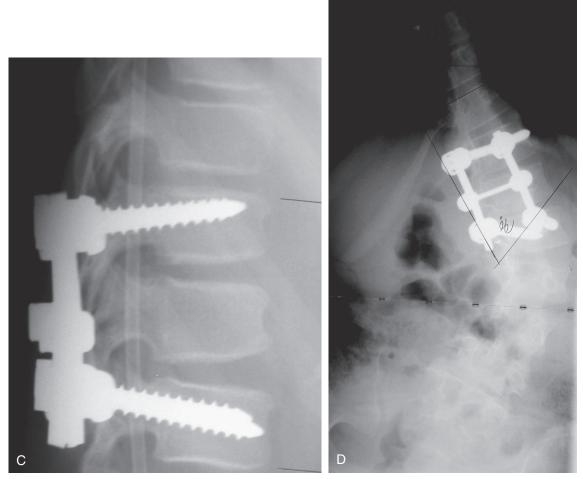
Continued

Fig. 47-2 cont'd *(C)* and was found to have dural rupture intraoperatively. Approximately 18 months following surgery, the patient presented with post-traumatic spinal deformity *(D),* which was treated with positerior spinal fusion *(E).*

excellent bony detail and soft-tissue detail, respectively. It is important to remember that by definition, patients with SCIWORA have no evidence of injury on conventional radiographs or CT imaging. In the absence of specific findings on conventional radiographs and CT in a patient with a suspected spinal column injury or neurologic deficit, most authors advocate the use of MRI to image the spine.[36–38] MRI can be helpful in ruling out spinal cord compression, occult fractures, subluxation, disk herniation, and/or presence of ligamentous injury. In addition to these roles, MRI in cases of SCIWORA can be used to define the location, type, and degree of neural injury.[39] The MRI findings in patients with SCIWORA can be classified as either extraneural or neural. Extraneural injuries most commonly involve the intervertebral disk, ligaments, and muscles, whereas neural injuries can be localized to the spinal cord. When evaluating the MRI images, the clinician or radiologist should pay special attention to the evaluation of the sagittal and axial T2-weighted and fat-suppressed T2-weighted or short tau inversion recovery (STIR) images for areas of abnormally increased signal intensity.[39] These

images best demonstrate the edema and hemorrhage associated with soft tissue and neural injury. With the increased availability and use of MRI and the increasing number of reports of patients with SCIWORA in whom MRI has been obtained, almost every soft tissue structure that provides stability to the spinal column has been shown to be injured in patients with SCIWORA.[23,37] These structures include the intervertebral disk, anterior longitudinal ligament, posterior longitudinal ligament, interspinous and other ligaments, and tectorial membrane.[21,22]

The neural injuries seen on MRI in patients with SCIWORA are most commonly seen as a region of abnormal signal intensity within the spinal cord. This signal change is most commonly seen on the T2-weighted images and generally represents edema or hemorrhage within the spinal cord. More specifically, the various patterns of abnormal signal intensity within the spinal cord commonly represent the various forms of extravasated hemoglobin including oxyhemoglobin, deoxyhemoglobin, intracellular methemoglobin, extracellular methemoglobin, and hemosiderin.[23,40,41] Gradient echo pulse sequences are even more sensitive to the

Fig. 47-2 cont'd *(E).*

presence of paramagnetic substances such as deoxyhemoglobin and may demonstrate small areas of acute hemorrhages as focal regions of very low signal intensity.[37] Careful evaluation of the spinal cord in all available planes should be performed in an effort to determine the percentage involvement of the transverse cross-sectional area of the cord. Grabb and Pang[37] have suggested that MRI evidence of major cord hemorrhage with involvement of most of the transverse cross-sectional area of the cord or cord transection is associated with permanent and complete cord injuries, whereas MRI evidence of edema or hemorrhage involving less than 50% of the cross-sectional area is associated with good recovery but residual deficits.

TREATMENT

The treatment of a pediatric patient with a spinal cord injury, regardless of the underlying injury pattern (i.e., vertebral column fracture, fracture with subluxation/dislocation, subluxation/dislocation alone, and SCIWORA), begins after the diagnosis is confirmed with appropriate imaging including conventional radiographs, MRI, and

often CT. Initial treatment is directed toward management of the airway, breathing, and circulation. The management of a neurologic deficit may include the administration of high-dose corticosteroids if the injury is diagnosed within 8 hours of its occurrence. Management with corticosteroids remains somewhat controversial and the current recommendation in children is based on the National Acute Spinal Cord Injury Study III (NASCIS III) trial in adult patients.[42,43] Management of vertebral column injuries (fracture alone, fracture with subluxation/dislocation, and subluxation/dislocation alone) is variable and is based on the magnitude and mechanism of the injury. Although the treatment strategies are covered in detail in other chapters, the basic considerations for surgical treatment are the need to decompress the spine and the neural elements and to stabilize the spine to prevent further injury.[14] The current recommendation for the great majority of pediatric patients with SCIWORA is nonoperative and may include corticosteroids and rigid external immobilization.[6,23,28,44] Surgery may be considered for patients with objective evidence of spinal cord compression or spinal instability resulting from an extraneural injury.[23] In a recent meta-analysis of 392 cases of SCIWORA that were reported in 80 manuscripts, Launay et al.[28] found that the spinal injury occurred in the cervical spine in 74% of the patients and in the thoracic spine in 26% of the patients in whom the level of injury was available. Furthermore, the authors found that the treatment protocols and outcomes were available for 98 pediatric patients; of those, 41 were treated with rigid immobilization for 8 weeks and 52 were treated with rigid immobilization for 12 weeks. Based on the outcomes in these patients and the rate of development of recurrent SCIWORA, the authors concluded that rigid immobilization for 12 weeks rather than 8 weeks results in improved outcomes ($P = .01$).[28] With the increasing availability and reliability of MRI, however, it is now possible to evaluate the extent of soft tissue damage and determine the length of immobilization needed according to the surgeon's best judgment. Obviously, close radiographic follow-up is needed, especially if shorter periods of immobilization are used.

PROGNOSIS

Pediatric patients with spinal cord injury appear to have a higher rate of neurologic improvement than adults with similar injuries.[3-7,15,16,35,45–48] Osenbach and Menezes[6] found that neurologic outcome was dependent on the severity of the initial injury. In their series, of the 37 patients with complete spinal cord injury, only 11% (4 of 37 patients) demonstrated partial motor recovery and one became ambulatory with assistance, whereas children with mild to moderate injuries recovered normal or nearly normal function.[6] Hamilton and Myles[5] also found that prognosis was based on the severity of the initial injury: of the
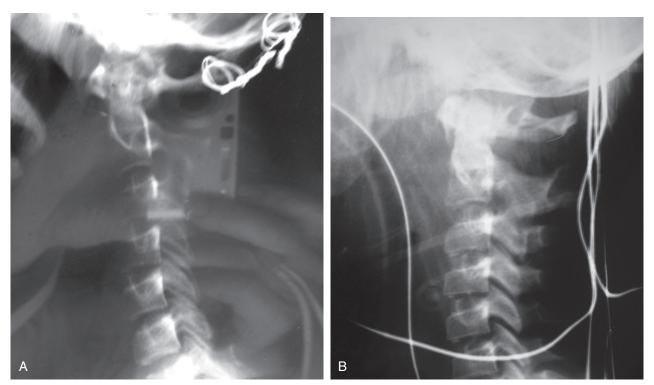
Fig. 47-3 Lateral radiographs *(A)* demonstrating atlantooccipital dissociation treated with posterior spinal fusion *(B).* The patient was nearly neurologically intact on pres-entation, and she made a full recovery.

patients with complete injuries, only 10% had partial motor improvement (one or two Frankel grades) and the remainder remained unchanged. In contrast, 74% of patients with an incomplete injury had significant improvement, and 59% had complete neurologic recovery.[5] Hadley et al.[7] noted that 20% (4 of 20 patients) with complete injuries demonstrated some level of recovery. Similar results
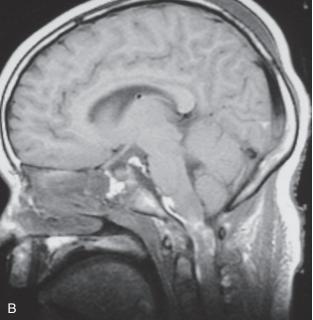
Fig. 47-4 Lateral radiograph *(A)* and sagittal MRI *(B)* demonstrating C1-C2 disruption with spinal cord injury. The patient had a complete injury at the C2 level and was ventilator-dependent quadriplegic. The patient was treated with a halo and never had any neurologic recovery.

Fig. 47-4 cont'd *C,* She presented 3 years following injury with post-traumatic spinal deformity.

have been reported by other authors.[3,4,15,16,35,45–48] As in all patients with spinal cord injury, the prognosis of patients with SCIWORA correlates well with the neurologic status on initial evaluation.[6,7,21,22,28] Patients with complete neurologic injuries only rarely improve, those with severe but incomplete lesions improve with time but are unlikely to regain normal function and those with mild to moderate neurologic deficits have the best chance of full recovery.[23] As discussed above, Grabb and Pang[37] have shown that the cross-sectional area of involvement of the spinal cord also predicts the degree of neurologic recovery. Although pediatric patients have a higher rate of neurologic recovery than adults, they must face many of the same complications associated with spinal cord injury such as spasticity, post-traumatic deformity, decubitus ulcers, infections associated with the need for ventilatory support and chronic bladder dysfunction, and psychological implications of living with a spinal cord injury.[14]

CONCLUSION

Each year, approximately 10,000 people in the United States present with a spinal cord injury,[1,2] but pediatric spine injuries account for only between 1% and 11% of these injuries.[3–19] Anatomic and biomechanical differences between children and adults lead to different patterns of injury than is seen in adults. For example, injuries such as atlanto-occipital dissociation, vertebral endplate fractures, and SCIWORA occur much more often in children than in adults. The unique nature of pediatric spinal injuries, combined with their relatively rare occurrence, make the detection and treatment of spine injuries in children more challenging, and therefore, one must always be vigilant when dealing with pediatric spine injuries to make the correct diagnosis and render the proper treatment.

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The Use of Cervical/ Thoracolumbar Orthoses, Halo Devices, and Traction in Children

INTRODUCTION

In 1975 the American Academy of Orthopaedic Surgeons (AAOS) defined an orthosis as an external device that is applied to the body to restrict motion in a body segment. The first recorded use of orthotic devices to immobilize the spine can be found in the chronicles of the fifth Egyptian dynasty. Galen (131–201) was the first physician to advocate braces for the correction of spinal deformities. Nicolas Andry (1658–1742) designed an iron cross immobilizer for the cervical spine. Throughout the 19th century, the Europeans developed a large number of devices fashioned from steel, leather, and plaster for the correction of spine deformities. The modern era of orthotic treatment for spinal deformities began with the development of the Milwaukee brace (cervicothoracolumbar sacral orthosis [CTLSO]) by Blount and Schmidt.[1]

Halo devices with rigid connection to the skull may allow greater immobilization of the cervical spine which has been rendered unstable as a result of tumors, inflammatory and degenerative diseases, congenital malformations, and trauma. Traction, which may be used in conjunction with an orthosis, is used for reducing rotatory deformity of C1 on C2, for fracture dislocations of the cervical spine, and maintaining alignment when the spine is unstable.

CERVICAL ORTHOSES

Orthotic devices (orthoses) are generally named after the body regions that they span. For example, a cervical orthosis (CO) spans the cervical region, whereas a CTLSO covers the entire length of the spine. COs can be divided into two groups: COs and cervicothoracic orthoses (CTOs). These devices may also be grouped as soft, reinforced, and rigid. COs restrict the motion of the cervical spine to prevent pain, provide presurgical and postsurgical spinal stability, and provide emergency protection immediately following trauma.[2] In the past few decades, the soft foam cervical collar has evolved into a variety of COs using combinations of rigid and flexible materials to restrain cervical motion. Following soft tissue injury to the cervical spine, COs may restrict the motion of this region. This allows tissue healing by reducing the demand on the cervical muscles, and may also reduce pain by avoiding extremes of motion.

Typically, severe cervical spine injury can be treated with a halo vest orthosis or with spinal surgery and then a halo vest orthosis.[3] After several weeks to months, when there is sufficient osseous and ligament integrity of the traumatized cervical spine, the halo vest orthosis is removed and a less obtrusive orthosis applied (e.g., Philadelphia collar, soft collar, semirigid plastic collar). When an orthosis must be worn for an extended time, comfort is naturally an issue. In this particular situation, the soft and semirigid collar types of COs may be considered, understanding the compromise in stability associated with less rigid devices.

Sandler et al.[4] compared the amount of motion allowed by the foam collar, Philadelphia collar, Philadelphia with thoracic extension, and sterno-occipito-mandibular orthosis (SOMI) with the amount of unrestricted neck motion in normal subjects. All orthoses restricted motion to some extent. Generally, the collars ranked (from least restrictive to most restrictive): soft, Philadelphia, Philadelphia with extension, and SOMI brace. No collar restricted the motion of any of the subjects to less than 19 degrees of flexion-extension, 46 degrees of axial rotation, or 45 degrees of lateral bending, and most subjects demonstrated significantly more motion. However, the SOMI remains the orthosis of choice in levels C4 and above, where flexion control is critical. Below C4, the orthosis of choice is a CTO when extra leverage is needed.

CERVICAL ORTHOSES

The foam collar is an example of a soft collar (Fig. 48-1). It is usually encased in a knitted material and closes in the back using Velcro. As mentioned previously, foam collars provide very little support or control. They are usually used for the mildest of conditions such as whiplash-type injuries, for which they may provide comfort and proprioceptive feedback to help "remind" the patient to restrict motion voluntarily.

The Philadelphia collar is the best-known example of a reinforced cervical collar (Fig. 48-2). It is made of Plastazote reinforced with plastic anteriorly and posteriorly. They are sized taking into consideration the size of the neck and the distance between the chin and chest. The greater this distance, the more neck extension given. The pediatric collar has a tracheotomy opening, which allows for access to respiratory equipment if needed.

The Aspen collar is available in a pediatric version that is not a scaled-down version of an adult collar but is designed to fit the different occipital and mandibular shape of the pediatric patient (Fig. 48-3). This design increases immobilization and comfort as compared with the other pediatric collars, which are scaled down versions of adult braces.

Fig. 48-1 Foam collar.

CERVICOTHORACIC ORTHOSES

CTOs use circumferential supports to achieve attachment between the chin and occiput fixation. They can have from two to four rigid uprights to increase their stiffness. Examples of CTOs include the Minerva brace (Fig. 48-4) and SOMI (Fig. 48-5). When compared with collars, these devices improve control in all planes.[4] However, they are often less comfortable for the patient. The SOMI is a reinforced CTO and has three components: a sternal yoke, an occipital component, and a mandibular component. These are relatively easy to fit and are available in pediatric sizes. Johnson et al.[5] reported a 32% to 61% restriction of normal flexion-extension per level from C1-C2 to C7-T1 with this orthosis.

THORACOLUMBAR ORTHOSES

For nonoperative pediatric thoracolumbar injuries the orthotic treatment is the same as for adults. Because children tend to heal more quickly than adults, the treatment length is greatly reduced. For stable thoracolumbar fractures involving the anterior and/or middle columns, treatment with a custom-molded bivalved thoracolumbosacral orthosis (TLSO) is adequate (Fig. 48-6). This may help reduce the segmental angulation of the kyphosis and unload the force on the fractured portion of the vertebrae.[6] Secondarily, TLSOs reduce the segmental and gross motion of the spine. For posterior and middle-column injury through bone, the same mechanism applies and application of a TLSO may reduce the fracture and allow healing of the defect.

The Jewett hyperextension brace uses a three-point pressure system with one posterior and two anterior pads (Fig. 48-7). The anterior pads place pressure over the sternum and pubic symphysis. The posterior pad places opposing pressure in the midthoracic region. The posterior pad attempts to hold the spine in an extended position. It has a lightweight design that is more comfortable than the TLSO brace. Pelvic and sternal pads can be adjusted from the lateral axillary bar where they attach. No abdominal support is provided with this device. This also adds comfort as there is no abdominal compression. The Jewett brace can limit flexion and extension between T6-L2/L3 but is ineffective in limiting lateral bending and rotation of the upper lumbar spine. Indications for use of the Jewett brace include symptomatic relief of compression fractures and immobilization after surgical stabilization of thoracolumbar fractures. Contraindications to use the Jewett brace include three-column spine fractures involving anterior, middle, and posterior spinal structures and compression fractures above T6 because segmental motion increases above the sternal pad.

In general, ligamentous Chance fractures of the posterior and middle columns and severe burst and three-column injuries such as fracture/dislocations are generally too unstable to be successfully treated with an orthosis. Orthotic treatment of thoracolumbar injuries run the risk of deformity

Fig. 48-2 A child with a Philadelphia neck brace as seen from the front *(A)* and rear *(B).*

Fig. 48-3 An Aspen cervical orthosis.

progression, particularly if there is noncompliance with brace wear. It is important to relay the possible ramifications to the patient and the parents and to make certain that wearing parameters are met.

HALO DEVICES

Since the introduction in 1959 by Perry and Nichol, halo skull traction has become a universal and effective method for cervical spine stabilization in adults and children.[7] This device was first used in the spine to immobilize the cervical spine in poliomyelitis patients with paralytic cervical muscles. The halo has subsequently been used to provide stability of the cervical spine in cases of tumors, inflammatory and degenerative diseases, congenital malformations, postoperatively following surgical fusions, and trauma.

In a cadaveric study, Richter et al.[8] reported that the halo vest was superior to the soft collar, prefabricated Minerva brace, and Miami J collar for immobilization of the unstable cervical spine. All three semirigid devices reduced the range of motion (ROM) at both C1-C2 and C2-C3 of the intact spine significantly but did not provide as rigid immobilization as the halo device. When an odontoid osteotomy was performed to produce an unstable C1-C2 segment, the soft collar did not provide any clinically relevant stability to the region. The Miami J and Minerva

Fig. 48-4 A Minerva brace, anterior *(A),* lateral *(B),* and posterior *(C)* views.

Fig. 48-5 Sterno-occipito-mandibular orthosis (SOMI), front *(A),* rear *(B),* and lateral *(C)* views.

Fig. 48-6 Thoracolumbarsacral (TLSO) orthosis, lateral *(A)* and posterior *(B)* views.

braces provided moderate control in the sagittal plane, but allowed better control of "torque" in the upper cervical spine. The halo vest did not allow measurable motion in any plane with experimental external loading. The authors concluded that the halo with vest seemed to be the first choice for nonoperative treatment of unstable injuries of the upper cervical spine.

Although numerous improvements have been made to the various components of the halo device, the overall design principle remains the same. A ring is attached to a vest by four connecting rods (Fig. 48-8). Newer rings are made of composite materials which have the beneficial properties of being lighter in weight, radiolucent, and compatible with magnetic resonance imaging (MRI). Other improvements have allowed the fabrication of adjustable rings, convertible tong-to-ring designs, open rings or crowns (tiaras) that encircle only a portion of the head, and noninvasive designs (Fig. 48-9). Advances in plastic technology have led to the development of lightweight, durable, yet adjustable and quick-to-apply vests, which have replaced plaster casts. Cross-straps and supports stabilize and decrease shear stresses between anterior and posterior portions. Low-profile designs

for the uprights and connecting rods provide a manageable and more patient-friendly frame. Current plastic vest and connecting rod systems allow cervical spine adjustments in virtually any plane. Safety-knurled adjustment knobs, two-point flexion-extension supports with ratchets, and light-weight metals have allowed fine adjustments for fracture alignment.

Despite the advantages provided by these prefabricated vests, they do not always fit adequately, especially if limited sizes are available. If a vest fits poorly in a child, use of a form-fitting body cast or custom-molded, plastic halo vest should be considered. This is particularly true if there are skin problems or multiple injuries that require custom fitting to the trunk. Gortex-lined fiberglass casts are more durable, and the Gortex liner allows them to get wet.

Very few changes have been made to the design of the halo pin since it was originally manufactured. A pointed bullet-type pin with a broad shoulder may provide more rigidity at the pin-bone interface. Break-away torque wrench handles for the pins designed for one-time use have been introduced. These wrenches are designed to break off at a specific torque (e.g., 8 in-lbs, 0.90 N-m) and potentially save

Fig. 48-7 The Jewett hyperextension brace.

time. Although these are accurate, rechecking the pin torque with a calibrated torque screwdriver is prudent, particularly after multiple uses.

PIN INSERTION TECHNIQUES

PIN-SITE SELECTION

The preferred sites for halo-pin insertion have been evaluated in previous studies.[9–14] The optimal position for the anterior halo-pin placement, based on anatomic structures at risk and skull thickness, is the anterolateral aspect of the skull, approximately 1 cm superior to the orbital rim, caudal to the greatest circumference (equator) of the skull, over the lateral two thirds of the eye. This area is sometimes referred to as the "safe zone." Placement of a pin above the supraorbital rim prevents displacement or penetration into the orbit. Placement of the pin below the greatest skull diameter prevents cephalad migration of the pin.[9,12–15]

On the lateral aspect of the safe zone lies the temporalis muscle and fascia. Avoidance of this muscle by the halo-pin can reduce the amount of pain experienced from the procedure and is less likely to impede mandibular motion during mastication. In this area the bone is also very thin, making skull penetration, or pin loosening, more likely. Although there is cosmetic advantage of pin placement in the temporlis region behind the hairline, the anatomic and mechanical disadvantages of this site do not seem to warrant this location if other areas are feasible.[10,13,16]

Along the medial border of the safe zone lie the supraorbital and supratrochlear nerves and underlying frontal sinus. Insertion of pins lateral to the medial one third of the orbit avoids injury to these nerves and risk of penetration to the frontal sinus. [10,13,16]

Placement of pins on the posterior aspect of the skull is less critical. In this region, vulnerable neuromuscular structures are lacking, the skull is thick, and the bony contours are more uniform. For pin placement, the 4 o'clock and 8 o'clock positions are optimal.[9,12–15]

ANGLE OF PIN INSERTION

Studies have shown that perpendicularly inserted pins have superior fixation compared with those placed at 15 or 30 degrees to the skull surface. The explanation is that broader pin-bone interface with increased contact area is

Fig. 48-8 A 2-month-old child with an invasive halo.

achieved by placing pins perpendicularly. With any angulation, the shoulder of the pin may intercept the skull's outer cortex before the tip is fully seated.[9,17]

SKIN INCISIONS

The clinical advantage of making skin incisions prior to pin placement vs. direct insertion of the pin into the skin has not been demonstrated.[10] Loosening, infection, comfort, and resultant scars were not different by creating a skin incision before inserting pins. Skin incision can, however, cause problems with bleeding, which can momentarily delay the halo application procedure.

PIN INSERTION TORQUE

In children the recommended pin application torque should be between 2 to 6 in-lb depending on the age of the child.[7] In patients younger than 3 years, a multiple-pin (10 to 12), low-torque technique has been recommended to allow a greater range of pin placement and distribution of forces to the immature skull. In this age group the skull is too thin (weak) to accept high torque forces. As the skull thickens with age fewer pins with greater insertion torque may be sufficient (Table 48-1).

APPLICATION TECHNIQUE

PREPARATION AND SELECTION OF EQUIPMENT

The equipment required for the pediatric population and techniques of application are similar to those for older patients. Because of the small size of pediatric patients and infrequent need, manufacturers may not always carry a full inventory of parts. Custom-made components might be required. It is important that vest sizes and all material and equipment are inspected before starting the procedure. At least three persons are required for safe and effective halo application.[15] Positioning pins and mechanical head holders are helpful. The person holding the child's head should be aware of the fracture and be comfortably situated while maintaining the unstable cervical spine and halo in position. This task should not be left to an inexperienced member of the team.

APPLICATION OF THE HALO RING

In younger children custom rings may be required with closely spaced holes for multiple pins per quadrant. The halo ring can be applied under general anesthesia in younger children but preferably under local anesthesia in older patients so that they can "splint" their necks with voluntary muscle control and warn the physician of any change in neurologic status. Mubarak and colleagues[7] have outlined the use of custom halo devices in young patients. The halo ring is usually made 2 cm larger in diameter than the wire impression used to size the head. Ten to 12 standard halo skull pins can be used. The ten pins should be inserted to two-finger tightness or to a torque of 2 in-lb, avoiding the temporal region and frontal sinus. Once the halo is secured, manual traction on the ring can be used to control the cervical spine. Areas of tented skin surrounding the pins can be released with a scalpel.

APPLICATION OF THE VEST

Next, the vest and the suprastructure are applied. With continued manual traction, the patient's trunk is elevated to allow placement of the posterior part of the vest. The anterior half of the vest is placed, and the head and neck are positioned and the bolts secured. CT scans and skull radiographs can be obtained to visualize suture lines or bone fragments that are occasionally found in congenital malformations and following trauma.

Pin care in children is identical to that for the adult patient. Postoperatively, the halo-pin sites should be cleaned daily, or as needed, with hydrogen peroxide or antiseptic solution. The hair and scalp should be washed at least once a week. If the pin sites become infected, oral or parental

Fig. 48-9 A noninvasive halo device, front *(A)* and lateral *(B)* views.

TABLE **48-1**   Authors' Preference for Number of Pins Inserted and Torque Used During Halo Ring Application in Children

AGE (YR)	NUMBER OF PINS	TORQUE (POUNDS)
0–3	10–12	2
4–7	8	4
8–11	6–8	6
12	4	8

antibiotics should be started. If drainage persists in spite of antibiotics, removal of the infected pin is necessary.

COMPLICATIONS

As in any other surgical procedure, substantial complications can occur following application of the halo skeletal fixator. Pin loosening and pin-site infection are the two most common complications.

PIN LOOSENING

Some studies have reported that pin loosening occurs in 36% to 60% of patients.[18–20] If a pin becomes loose in the absence of infection, the loose pin and remaining pins can be retight-ened once, as long as resistance is met within the first few complete rotations of the pin. If no resistance is met, the pin should be removed after placing a new pin in an adjacent location.[18] Placement of a new pin before removal of the loose pin will help maintain ring/crown fixation during pin change.

INFECTION

Pin-site infection has been reported to occur in 20% to 22% of patients.[18–20] If drainage or erythema develops at a pin site, management should include bacterial cultures, appropriate antibiotic therapy, and more "aggressive" local pin care. If there is no response, as suggested by continued drainage, development of cellulitis, or abscess, the pin should be removed after insertion of a new pin at a different site. Incision and drainage of the abscess should then be performed, cultures obtained, and parental antibiotics continued.[18]

PIN-SITE BLEEDING

This is a rare complication, occurring in only 1% of patients.[20] It is more likely to occur in children with bleeding disorders. Pin-site packing may be used in an attempt to stop bleeding.

DYSPHAGIA

Difficulty in swallowing has been reported in 2% of patients.[20] This complication tends to occur if the head and neck are hyperextended. Repositioning the halo with less cervical extension may relieve the problem if this can be done without compromising the cervical reduction.

DURAL PUNCTURE

This rare but potentially serious problem usually occurs after significant trauma to the halo. Examples include a forceful blow, or a fall, onto the device.[15,16,18] Initial symptoms of a dural puncture may include headache, malaise, visual disturbance, and other local or systemic symptoms.[21] Leakage of clear cerebrospinal fluid around a loose or deeply seated pin should alert one to the possibility. Radiographs may reveal previously unnoticed skull fractures. Special tangential radiographs obtained perpendicular to the pin may demonstrate pin penetration through the inner cortex of the skull.

Treatment of dural puncture from a halo pin consists of hospitalization, prophylactic antibiotics, and pin removal after placement of a new pin at an adjacent site. Elevation of the head of the bed decreases intracranial fluid pressure and reduces leakage. Dural tears usually heal in 4 to 5 days. Surgical exploration and dural repair is necessary for leaks that do not respond to conservative treatment.[16]

Other complications associated with the use of halo devices that have been reported include residual neck pain or stiffness, decreased cervical rotation or lateral bending capabilities, and suboptimal scar healing.[18]

TRACTION

Commonly used traction methods in pediatric trauma include use of halter, Gardener-Wells tongs, and the halo apparatus.

HALTER

In this method, traction is applied with one limb of the halter around the mandible and the other around the occiput, with weights exerting a direct pull over the edge of the bed. This simple device is used mainly in reducing a rotatory deformity of C1 on C2 in atlantoaxial fixation when other measures have been unsuccessful.[22–24] Halter traction can be uncomfortable for children because of irritation caused by the straps on the facial skin. If prolonged traction is required, the halter may have to be replaced by halo traction. Depending on the size and age of the child, 5 to 8 pounds of weight is the most that can be tolerated for any length of time. Halter traction is not appropriate for traumatic forms of cervical spine instability.

GARDENER-WELLS TONGS

Crutchfeld first described in 1933 the use of tongs for the treatment of fracture-dislocation of the cervical spine. Gardener-Wells tongs have become the most widely used device. Its use is restricted to the adolescent and adult patient rather than the child. Although the tongs allow more weight to be applied than when a head halter is used, they do not confer as much stability as the halo device and cannot be readily incorporated into a vest or plaster jacket. Lerman et al.[25] reported that the pullout strength of tongs tightened to the manufacturer's recommended level decreased with increased use. This was partly attributable to spring and/or pin wear and suggested that heavily used tongs be replaced or recalibrated.

Gardener-Wells tongs can be applied under general or local anesthetic. The pins should be placed inferior to the equator of the skull in line with or 1 cm posterior to the external auditory canal just above the pinna. Insertion of the pins anterior to the auditory canal will extend the head with traction, whereas flexion of the head occurs if the pins are placed posteriorly. After 24 hours, the pins should be retightened so that the indicator is flush with the spring-loaded pins.

CERVICAL SPINE TRAUMA

Traction is one of the oldest methods used for correction of spinal deformity and has remained a mainstay of treatment for cervical spine trauma. Children younger than 10 years most commonly have cervical spine trauma above C3 and children older than 10 years more typically have cervical spine injuries below this level. If reduction of a fracture-dislocation is attempted with traction, the patient must be monitored closely radiographically and neurologically during application of traction.[26] No more than 2- to 3-pound increments in younger children should be used. The most important complication to avoid in treating these patients by this method is overdistraction. Ten minutes after the application of each increment a lateral radiograph should be taken; if there is any overdistraction at the fracture or dislocation site, weight should be removed and a repeat radiograph taken. For fractures from the occiput to C2, only 2 to 5 pounds of weight is required to obtain safe reduction. For fractures below C2, 5 pounds of weight usually must be added for each level. This weight is applied at 10-minute intervals, after which a lateral radiograph should be taken and a neurologic examination performed. If there is more than 7 to 10 mm distraction at the fracture or dislocation site, or change in neurologic function, the weight should be decreased and the examination repeated.

ATLANTO-OCIPITAL DISLOCATION

Disassociation between the skull and C1 is often fatal and tends to be a distraction injury.[27–29] Because of the extensive ligament disruption that occurs with this injury, any traction

should be applied with extreme caution and is often contra-indicated. A halo vest should be applied, or surgical stabilization performed, as soon as the child is medically stable because of the high risk of overdistraction. In the event of surgery, the halo vest can be maintained postoperatively to aid stability.[30–32]

ODONTOID FRACTURE

Plaster casts and cervical braces are effective for type I fractures and epiphysiolyses of the odontoid process.[33] In children, reduction of odontoid fractures can be aided with halo traction. Extension and posterior translation often helps reduce the fracture. Thereafter, the halo can be incorporated into a jacket or vest. Surgical intervention is usually unnecessary.[33–35]

C2 FRACTURE

Traumatic spondylolisthesis of C2, or "hangman's fracture," can be reduced with extension of the cervical spine combined with gentle traction. Eight to 10 weeks of immobilization in a Minerva jacket or halo vest is required.[36]

C3-C7 FRACTURE

These fractures occur more commonly in children older than 10 years.[37] The main use of skeletal traction in these injuries is for fracture/subluxations or dislocations of one vertebra on another. Traction can be applied with Gardener-Wells tongs or a halo, as described previously. During the procedure, the patient must be monitored carefully both radiographically and neurologically.[26] For reductions done under a general anesthetic spinal cord monitoring is recommended.[37]

COMPLICATIONS

Complications as a result of traction are not uncommon. Meticulous attention to detail in the application of the traction device and close clinical observation of the patient in traction will lessen the likelihood of these complications.

NEUROLOGIC

Spinal traction can result in injury to the spinal cord, cranial nerves, brachial plexus, or peripheral nerves. The spinal cord is most likely to be injured in children with cervical spine injuries. The skull should not be hyperflexed and overdistraction should be avoided.[26]

Brain abscess after insertion of skull traction is a rare and serious complication.[38] It may be suspected in a patient who has onset of headache, fever, focal neurologic signs, or change in mental status. If the dura has been perforated cerebrospinal fluid may be seen around the offending pins. Adequate preventive measures include proper sterile dressing and daily care.

The abducens (VI) cranial nerve has a long intracranial course over the petrous temporal bone and can be injured during skeletal traction using halo devices and Gardener-Wells tongs.[39] The theorized mechanism of injury to the abducens nerve involves stretch or traction force, which causes localized ischemia or a change in nerve position. There is loss of lateral gaze and diplopia. Most cases of abducens nerve palsy tend to spontaneously improve with reduction in traction force. The lower cranial nerves can also be stretched where they exit the skull.

Excessive traction can result in brachial nerve palsies. The most commonly involved nerve roots are C6, C7, and T1. C6 and C7 have a near vertical direction which increases the susceptibility to traction neuropraxis. T1 is vulnerable because of its course over the first rib. Monitoring cranial and cervical roots is important during periods of traction.

DERMATOLOGIC

Pressure sores, especially over bony prominences, can become a problem in a patient who has to be recumbent in traction for a long time. This situation can be more difficult in a child that has lost sensation to the skin and who also might be unable to communicate that he/she feels pain. To prevent bedsores, beds should have foam on top of the mattress. Extra padding or a "donut" cushion should also be placed over bony prominences. If possible, the patient should be rolled every 2 to 4 hours. The child's back should be regularly inspected for red areas that may forewarn of a pressure sore. In the event of skin breakdown, additional pressure-relieving measures should be carried out, including consideration to halo-wheelchair traction, if possible. Rotatory beds should be used if traction is anticipated for more than 24 hours.

VASCULAR

Deep vein thrombosis is an uncommon complication in children. Leslie et al.[40] reported a 3.7% incidence of thrombosis in 54 children undergoing halo-femoral traction for an average of 28 days. There were no clinically silent cases.

Superficial temporal artery laceration has been reported following use of Gardener-Wells tongs. This produced chronic recurrent episodes of pulsatile bleeding from the pin site and necessitated arterial ligation to control the bleeding.[41]

CONCLUSION

Orthoses for spinal injuries function to provide biomechanical stability and protect the spinal column from loads and stresses that may cause progressive angular and translational deformity. In both the cervical and thoracic regions, these devices restrict motion of the spine to decrease pain, provide pre-and postsurgical spinal stability, and emergency protection immediately following trauma. Unlike halo immobilization, traction devices

such as the halter and Gardener-Wells tongs do not confer the same degree of stability to the spine. They are useful for reduction of rotatory deformity of C1 on C2 subluxation and in other types of fracture-dislocation of the cervical spine when other measures have been unsuccessful. The use of these devices is not without risk and can result in complications. Appropriate patient and device selection, together with a comprehensive knowledge of all aspects of each device may lessen the likelihood of complications from occurring.

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The Use of Spinal Instrumentation in the Traumatically Injured Pediatric Spine

INTRODUCTION

Pediatric spine injuries are uncommon. However, they contribute to significant morbidity and mortality in children. Furthermore, the mechanism of injury, initial management and definitive stabilization of the pediatric spine vary considerably from the adult patient. Hence, it is important to have a thorough understanding of the developmental anatomy and biomechanics of the growing spine. This aids in anticipating potential pitfalls and adequately treating the long-term sequela of these injuries. Traditionally, many pediatric spine injuries were managed with external immobilization in a custom molded brace, collar, or halo vest.[1–4] However, compliance with these measures varies and the halo vest, in particular, has a high incidence of associated complications including pin-site[5] infection, loosening, scarring, osteomyelitis, and cranial nerve palsies.[6,7] Dormans et al.[6] reported a 68% complication rate in 37 children aged 3 to 16 years who were managed with a halo vest. With improved instrumentation and techniques, the indications for early internal fixation are expanding.

EPIDEMIOLOGY

It is difficult to know the true incidence of pediatric spinal trauma because children with injuries severe enough to cause spinal fracture may die from associated injuries.[8] Reddy et al. looked at 2614 pediatric patients reviewed, 84 sustained vertebral fractures and 50 had neurologic injury without radiographic abnormality. A total of 164 fractures were identi-

fied. The thoracic region (T2–T10) was most commonly injured[9] followed by the lumbar region (L2–L5) with 38 fractures,[9] the mid-cervical region with 31 fractures,[10] the thoracolumbar junction with 24 fractures,[9] the cervicothoracic junction with 13 fractures,[10] and the craniocervical junction with 11 fractures.[11] The entire cervical spine accounted for 55 fractures. The Trauma Audit Research Network Database spanning 1989 to 2000 includes 19,538 children younger than 16 years. Out of these children, 1.54% (301/19,538) suffered a cervical spine injury. Of these children, 21.9% sustained cervical cord injury and 7% manifested spinal cord injury without radiographic abnormality (SCIWORA). Both cord injury and SCIWORA was much more common in children younger than 8 years.[12] Patel et al.[13] found an incidence of cervical spine injury of 1.5% (1098/75,172) in the National Pediatric Trauma Registry. The mean age of the study group was 11 years and 61% were boys. As expected lower cervical spine injuries occurred mostly in children older than 8 years.[14] However, upper cervical injuries were prevalent in all age groups. Neurologic injury occurred in 35% of the children and was complete in 24%. SCIWORA occurred in 17% of injured children.[13] Except for those associated with birth trauma, children younger than 3 years rarely incur fractures below C2.

The mechanism of injury varies with the age of the patient. However, motor vehicle accidents cause the highest percentage of injuries. In children aged 0 to 5 years, violence and falls contribute the next highest number of injuries. In the age group 6 to 15 years, sports and violence are major culprits.[15] Diving is most commonly associated with cervical spine injury followed by football, gymnastics, and wrestling.[16] In neonates, the leading cause of cervical injury is birth trauma. MacKinnon et al.[17] reported that the injury occurred from C0 to C4 in 64% of patients. The majority of thoracolumbar fractures occur in children between the ages of 14 and 16.[18] The majority of injuries occur between T4 to T12 followed by L2.[2]

INITIAL MANAGMENT

As with any trauma, maintaining strict adherence to Advanced Trauma and Life Support (ATLS) protocol is vital for a successful outcome. A trauma patient is assumed to have

a spinal cord injury until proven otherwise. Thus, the patient must be immediately immobilized and transported carefully to avoid exacerbating neurologic injury. The head to chest ratio for young children is very high, and transportation on a regular backboard induces cervical kyphosis.[19,20] At age 8 years, the chest attains 50% of its adult size and this discrepancy normalizes. Until then, rolls placed under the chest or an occipital recess help maintain cervical alignment. The use of tape and sandbags provide superior immobilization of the cervical spine compared with a hard collar. In patients wearing a helmet, removal ideally occurs in the emergency department while the head is manually stabilized. One must always be cognizant of the ABCs of all emergency care, particularly because many children with cervical level injury have compromised respiratory function. Route of intubation, either orotracheal or nasotracheal, does not appear to cause neurologic injury, when performed in a controlled manner.[21] The role of steroids in the pediatric population remains unclear. Bracken et al.[22] reported methylprednisolone improved spinal cord recovery up to 5 motor points if administered within 8 hours of injury. Most treating physicians use methylprednisolone in children, although only 15% of patients in the study were between 13 and 19 years old.

The injured child should undergo an expedited, yet thorough history and physical evaluation as the incidence of associated injuries is very high.[23] Cardiac, abdominal, renal, and bladder injuries are often detected after the initial trauma evaluation. Patients with upper cervical injury may have cardiopulmonary arrest. Head injury occurs with spinal cord injury in 25% to 50% of patients.[19,24] On palpation of the spine, one must assess spinous process step off and tenderness as these may indicate underlying injury. Torticollis is another indication of cervical spine injury. The neurologic examination must be repeated at short, regular intervals to ensure the rapid detection of deterioration. It is important to determine the presence of transient neurologic symptoms. At the time of examination, the patient may not manifest any deficits; however, these patients may have occult instability, which if not detected may have catastrophic consequences.

CERVICAL SPINE INJURIES

ANATOMY AND BIOMECHANICS

The pediatric spine is more resilient to trauma than an adult spine. It has a greater capacity for growth and remodeling. The high water content of the immature spine allows more efficient loading of forces. This water content decreases from 88% at birth, to 80% at age 12, and down to 70% in old age.[8] An important feature in young children is the relatively large head size when compared with the body. Whereas in adults the fulcrum of motion is at the C5-C6 level, in infants the greatest motion is at C2-C3.[25,26] This helps explain the large proportion of upper cervical injuries seen in children younger than 8 years, after which the injury pattern resem-

bles that of adults. The large head size forces the cervical spine into a kyphotic deformity, which may worsen neurologic deficits. Thus, to maintain the neck in neutral position, either the torso must be elevated or the occiput placed in a cut recess.[19,20]

The pediatric spine demonstrates marked elasticity when compared with adults. The cadaveric spine of neonates can tolerate up to 4.4 cm of stretch without fracture; however, the spinal cord can only be stretched 0.5 cm. Thus, injuries that do not cause a fracture can still manifest marked spinal cord trauma. The flexibility of the pediatric spine derives from various factors. The neck muscles are underdeveloped and the tunicate processes underdeveloped, which leads to flatter articular surfaces.[27] The facet joints are shallow with a horizontal orientation, rather than oblique as found in adults, which allows translational mobility both laterally and in flexion/extension.[28] This is most noticeable in the upper cervical vertebrae. In addition, the ligaments and facet capsules permit significant stretching.[28] These factors in combination allow elasticity of the pediatric spinal column when compared with adults. The anterior wedging of vertebral bodies seen before the age of 12 years likely represents a ring apophysis that has yet to ossify, rather than a cause for ventral slippage between segments.[29]

RADIOGRAPHIC EVALUATION

Plain x-rays remain the initial screen to detect cervical spine injury. Multiple studies have attempted to stratify patients into low- and high-risk groups to identify those needing cervical spine radiographs. The National Emergency X-Radiography Utilization Study reported on 3065 children with 30 cervical spine injuries. The absence of all five of the following points had a negative predictive value of 99.9%: altered level of alertness, intoxication, posterior midline cervical spine tenderness, distracting painful injury, and focal neurologic deficit.[23] In this study, patients underwent standard three-view radiography: anteroposterior (AP), lateral, and odontoid. Of those patients who did not meet all five criteria, 1% had a cervical spine injury. Jaffe et al.[30] proposed obtaining cervical radiographs in any child who presented with neck pain or tenderness, limited neck range of motion, history of neck trauma, decreased sensorium, or neurologic deficit. The synchondrosis at the base of the odontoid does not fully ossify until age 7 years. This can result in difficulty distinguishing a fracture from normal development; thus, some authors advocate omitting the odontoid view.[31,32] In the study by Buhs et al.,[31] the odontoid view did not diagnose any of the injuries in the 10 patients younger than 9 years who had sustained a cervical spine injury between the occiput and C3. In practical terms, given the difficulty in obtaining the odontoid view in patients younger than 5 years, in appropriate patients an AP lateral x-ray is combined with a computed tomography (CT) scan of the occiput to C3 with reconstructions to fully evaluate the upper cervical area. When evaluating plain radiographs, one

must consider the most common normal radiographic variants in children. Pseudosubluxation in the upper cervical spine occurs as a normal variant. Cattell and Flitzer reported on 160 children and found 40% of children younger than 8 years harbored at least 3 mm of subluxation of C2 on C3. To delineate pseudosubluxation from a true injury, one draws a posterior intralaminar line, which is disrupted in the latter.[33] Other normal variants to consider include absence of cervical lordosis, anterior wedging of the vertebrae, and widening of the predental space.[33] In addition, the pediatric spine may lack cervical lordosis and contain pronounced vascular channels in ossification centers which appear as fractures.[34] Flexion and extension plain x-rays evaluate dynamic cervical spine stability. They are contraindicated in patients with neurologic deficit; these patients benefit from magnetic resonance imaging (MRI). The value of flexion and extension films in the acute setting is limited by muscle spasms and often is repeated several days later in the office setting. Some authors dispute the value of these dynamic x-rays, stating they add little in the setting of normal AP and lateral films.[35,36]

The role of CT scanning to diagnose cervical injuries in children remains limited. Children younger than 10 years often harbor ligamentous injury not readily appreciated on CT.[37] Thus, a normal CT scan cannot be used exclusively to rule out cervical spine injury. Fractures in the axial plane of the CT, such as Salter Harris I fracture through the odontoid synchondrosis, may not be detected without two-dimensional (2-D) reconstruction views. However, the value of CT scanning with reconstructions for surgical planning remains unquestioned.

The role of MRI will likely expand in the evaluation of children with cervical spine injury. MRI delineates the extent of damage, influences surgical approaches, and prognosticates likelihood of recovery. Furthermore, MRI has proven useful in the diagnosis of cervical spine injury. In a retrospective review of 52 children whose initial plain films and CT were negative, MRI detected 16 abnormalities. Six of these required either surgical stabilization or external fixation.[38] Similarly, Flynn et al.[39] identified 15 MRI abnormalities in 74 children with initial negative imaging. Frank et al.[40] advocate the use of MRI in obtunded patients for clearing the cervical spine because they found it effective and cost-efficient. As MRI continues to develop at a fast pace, the use of MRI in trauma patients will only increase.

TREATMENT STRATEGIES

Approximately 30% of cervical spine injuries require surgical intervention.[3,4] With regard to timing of surgery, most surgeons would agree urgent decompression remains probably provides the best chances of neurologic recovery.[41] There are several factors to consider in surgical decision making include anterior versus posterior approach, choice of graft material, and type of instrumentation. Preopera-

tive radiographic workup should include plain x-ray, CT, and MRI. Generally, the approach taken addresses the column of injury, with a combined anterior/posterior procedure for injury to all three columns. Plain films, CT, and MRI aid in determining the approach. The posterior surgical options include onlay fusion or wiring with external immobilization, lateral mass plating/screws, and pedicle screws (at least for C2). Anterior options include diskectomy and fusion with a plate and screws.

An important consideration for the surgeon is the choice between autogenous bone graft versus allograft. Allograft provides satisfactory fusion rates when used in anterior constructs under compression[42]; however, its failures are well documented in posterior constructs.[43] Rib, iliac crest, fibula, and calvarium are suitable options for autograft. Iliac crest bone grafting incurs considerable morbidity including persistent pain, hematoma, and infection.[44] In children younger than 3 years, the iliac crest may be cartilaginous and inappropriate for grafting.[45] Rib grafts provide an adequate source of bone but possess a relative lack of strength. Fibular grafts are ideal for long constructs. Associated morbidity with fibular grafts includes pain, postoperative tibial stress fractures, and motor deficits.[46] Autologous split thickness calvarial grafts appear to have the advantage of abundant graft, with decreased resorption, and less morbidity.[45,47] Further study with Bone Morphogenic Protein (BMP) in the pediatric cervical spine is required prior advocating its use. Certainly, the reports of swelling observed following its use in anterior cervical spine surgery in adults raises concerns.[48]

In children one must ensure the spine can accommodate instrumentation. In this respect the delineation of the osseous anatomy by CT is very helpful. In addition, one must consider the growth potential of the spine. At 10 years of age, the spine is near adult height; thus, surgery is less likely to induce progressive deformity. The epiphysis of the vertebral bodies contributes most of the growth potential, with little potential in the posterior elements. Thus, performing an anterior diskectomy and fusion will likely not lead to increased kyphosis. However, prior to age 5 years the vertebral bodies may not accommodate anterior plate and screws. In addition, specialized pediatric instrumentation with low profile and reduced screw lengths are available.[49] The long-term effect of cervical fusion in young children remains uncertain. The risk of adjacent segment degeneration appears similar to the 25% at 10 years reported in the adult literature.[50,51] Interestingly, McGrory and Klassen[51] found a risk of 38% of extension of fusion past the levels intended. This occurred within the first 2 years following surgery. Recently, absorbable cervical plates have been approved for use and shown good early clinical results.[52] Their potential in pediatric patients appears promising. Onlay fusion is probably the best option for patients prior to age 5 years as the lateral masses are likely too small to hold screws.

THORACOLUMBAR FRACTURES

ANATOMY AND BIOMECHANICS

From Bick and Copel[53] we know that the thoracolumbar vertebrae form from three main ossification centers. An ossification center forms each side of the neural arch and the body. The neurocentral synchondrosis, the junction between the ossification centers, is visible until 3 to 6 years. It can be misinterpreted as a congenital anomaly or fracture in younger children. Secondary centers of ossification occur in flattened, disk-shaped epiphyses superior and inferior to each vertebral body. These centers provide longitudinal growth but do not cover the entire vertebral body. Ossification of these growth plates at the age of 7 to 8 years creates the radiographic impression of a groove at the corner of each vertebral body. This groove is circumferential around the upper and lower end plates of each vertebra. The ligaments and disks attach to this groove, which is therefore an apophyseal ring. The ring apophysis develops its own ossification center by the age of 12 to 15 years and fuses with the remainder of the vertebra at skeletal maturity.[53]

CLASSIFICATIONS

There are no accepted classification systems for defining injuries to the pediatric thoracolumbar spine. Therefore, the classifications are extrapolated from our understanding of the skeletally mature spine.[54] The Denis model for the spine divides the spine into three columns: anterior, middle, and posterior.[55] Fracture stability is determined by the potential for mechanical and neurologic deterioration. As a general guideline, Denis' three-column model states at least two columns must be disrupted to lead to instability. White and Punjabi[56] suggested five specific indicators of spinal instability: greater than 33% canal compromise, translation of greater than 2.5 mm of vertebral bodies, vertebral body collapse with widening of pedicles, bilateral facet dislocation, and greater than 50% collapse of the anterior vertebral body.

A compression fracture results from failure of the anterior column with an intact middle column. A burst fracture occurs with involvement of both the anterior and middle columns. A seatbelt fracture is a compression injury to the anterior column, with distraction of the middle or posterior columns. In fracture dislocation all three columns fail.[55] Fractures of the lumbar vertebral apophysis can occur as long as it is weaker than the bony vertebral body or the annulus fibroses.[57] These can herniate into the spinal canal and present with back pain and neurologic deficits similar to a herniated disk in adults.

TREATMENT STRATEGIES

A majority of thoracolumbar spine trauma in the pediatric spine can effectively be treated with conservative management. However, the amount of wedging that will remodel is limited to less than 30 degrees.[58] The degree of retropulsion of the posterior wall of the vertebral body in to the canal will determine the degree of neurologic instability. CT and MRI provide a thorough appreciation of the extent of damage observed in a burst fracture. The degree of canal compromise is determined by measuring canal diameter above and below the injury and comparing to canal diameter at the level of the injury. Because the endplates are often involved these fractures can lead to a progressive kyphosis from the discrepancy between anterior and posterior column growth.

Transpedicular screw fixation has become an increasingly popular procedure in the treatment of not only spinal deformity but also trauma. Earlier posterior fixation methods typically involved the use of hooks and wires. Pedicle screw-assisted instrumentation, however, provides more rigid fixation. It allows fixation of the implant to all three columns of the spine. The addition of internal fixation serves the dual function of improving arthrodesis by rigid immobilization of the instrumented segments and correcting preexisting deformities by allowing application of greater corrective forces.[5] The screw-bone interface is generally stronger than the hook-bone and wire-bone interface. Another advantage is the fact that the posterior elements do not need to be intact. The hardware involved in posterior pedicle screw fixation is considered safe and effective for stabilizing a spinal motion segment.

Ruf et al. have described the use of pedicle screw instrumentation in patients as young as 1 year[59]. They performed a retrospective review of 19 consecutive operations in 16 patients with insertion of 91 pedicle screws for various spinal disorders in 1- and 2-year-old children. Short-term complications occurred in two patients (one pedicle fracture, one infection) and long-term complications in three patients (one screw breakage, two failures of screw connection). Three of 91 screws were misplaced without any neurologic symptoms. There are, however, several rare but significant complications that may occur with anterior or lateral cortical breach. In contrast to the well-described vascular and visceral complications associated with anterior thoracic instrumentation posterior, pedicle screws can be placed safely and with a low incidence of complications.[60]

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Evaluation and Surgical Management of Cervical Spine Injuries in Children

INTRODUCTION

The anatomical and biomechanical differences of the maturing spine produce substantial variation between the pediatric and adult populations with regard to clinical presentation, diagnosis, and functional outcome of cervical spine injuries. The more favorable outcome of pediatric cervical spine injuries in comparison to adults underscores the importance for timely diagnosis and management to prevent further progressive neurologic deterioration.[1,2]

Traumatic injuries of the cervical spine are relatively uncommon in the pediatric population. The incidence of pediatric cervical spine injuries has been reported to be between 1% and 9% of all cervical spine injuries.[3,4] However, they constitute the majority of all traumatic spinal injuries in children, up to 72%.[3]

The pediatric cervical spine has generally developed adult characteristics and is approximately adult size by 8 to 10 years of age.[3,5] Based on developmental and maturational differences in the pediatric cervical spine, it is appropriate to divide the population into two groups when assessing types and mechanisms of injuries: (1) younger children (9 years or younger) and (2) older children (older than 9 years).[3,6] Injuries occurring in the pediatric population younger than 2 years are extremely rare and are accounted mostly to birth trauma or child abuse.[6–8] Younger children predominately have injuries to the upper cervical spine, defined as occiput to C2.[6] These children are also more vulnerable to sustain ligamentous injuries than

fractures.[3,9] Neurologic injuries secondary to cervical trauma are most common in the younger group and are associated with a higher mortality rate.[3,10] Motor vehicle accidents (MVAs) remain the most common mechanism of injury regardless of the age of the child.[3,9]

Older children more closely follow the pattern of adult cervical spine injuries, most often involving the lower cervical spine.[3,6,10] Of 64 cases of cervical spine injuries seen at the Barrow Institute in pediatric patients older than 10 years, 42 of them (70%) had involvement of the lower cervical spine, below C2.[3] The occurrence of cervical spine fractures is much more common in older children.[3,7]

ANATOMIC FEATURES

The pediatric cervical spine is fundamentally different from that of the adult cervical spine because of the anatomical differences contributed to development.[3] These normal variants have led to missed diagnoses, especially in children with immature spines, at reported error rates of up to 24%.[4]

Younger children have greater ligamentous laxity leading to hypermobility of the spine in comparison to the more limited mobility of the cord.[8,11] The facets of the immature cervical spine lie considerably more horizontal than that of the mature spine.[5,11] These features of the cervical spine, along with the disproportionately large mass of the skull and the underdeveloped neck muscles, render the young child's upper cervical spine particularly vulnerable.[5,11] It is important to be aware that these features also can lead to a normal physiologic variation of the pediatric cervical spine, such as an increased atlanto-dens interval and pseudosubluxation, that would be considered abnormal if observed in the adult spine.[5]

Variations in the cervical spine growth centers may resemble fractures.[4,5] Many of these synchondroses do not completely fuse until 13 years of age.[5,11] The vertebral bodies of the cervical spine in younger children have a normal slight anterior-wedged shape, which can be misdiagnosed as vertebral body fractures.[3,4]

INITIAL EVALUATION AND MANAGEMENT

Any polytraumatized child should be managed and evaluated with a high index of suspicion of a cervical spine injury. Extreme precautions are mandated for any child who experiences any of the following: a significant mechanism of injury (i.e., MVA), a distracting painful injury, an altered mental status, focal neurologic deficits, head trauma, or midline cervical tenderness.[6,12] These patients need to be immobilized aggressively for protection from additional injury and evaluated thoroughly by radiographs, if clinically indicated, to clear the cervical spine.[12] Overlooked injuries of the pediatric cervical spine can have devastating neurologic consequences, often resulting in death or lifelong disability.[4,6,10]

The initial assessment begins with the ABCs of trauma resuscitation. Management of the airway should only commence with the assumption that a cervical spine injury is present. Until the cervical spine can be cleared clinically and radiographically, precautions should be used to immobilize the spine.[12] To protect against unwanted motion, a rigid collar should be used with additional enforcement of tape and sandbags.[12] The patient should stay in the collar until he or she is alert enough to participate with the physical examination.[12] In the patient that remains obtunded and intubated for more than 3 days, magnetic resonance imaging (MRI) has been shown to be effective, in addition to standard radiographs and computed tomography (CT), in ruling out cervical injury requiring continued stabilization.[13]

Because of the disproportionately large head in younger children, positioning them on a standard backboard produces an undesirable flexion of the cervical spine.[12] This flexion can lead to further anterior angulation or translation of the possibly unstable cervical spine. To avoid this unwanted flexion, the child should be placed on a modified spine board with either an occipital recess or a mattress pad to raise the torso[8,12] (Fig. 50-1). If this is not available, blankets may be used to build up the shoulders till roughly the external auditory meatus line up with the shoulders.[12]

The fact that 25% of children with cervical spine injuries present with no radiographic abnormalities emphasizes the critical importance of a thorough physical examination as the best means to a timely and accurate diagnosis of an injury.[4,5] First, the mechanism of trauma should be established, determining the likelihood of cervical injury.[5] The head and face should be carefully inspected for signs of trauma. Because of the disproportionate size of a child's head, as many as 66% of the pediatric cervical spine injuries are associated with head trauma.[1,3]

The physical examination should include a complete orthopaedic assessment. The neck should be inspected and palpated for obvious deformities, to elicit point tenderness, muscle guarding, or the presence of a step-off.[12] This should be followed by a complete neurologic examination assessing for sensory level involvement and voluntary movement.[12]
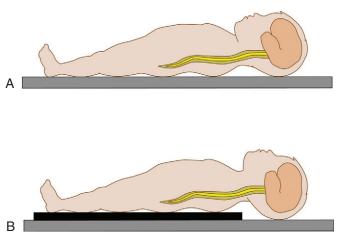

Fig. 50-1 When positioned on the standard backboard *(A)*, the disproportionately large head in younger children produces an undesirable flexion of the cervical spine with the potential of further displacement. Unwanted cervical flexion can be avoided by placing the child on a mattress to raise the torso *(B)*.

If the child has a normal neurologic examination, no tenderness or pain on active range of motion of the cervical spine, and no distracting injury such as a long bone fracture, then he or she is considered to have a stable cervical spine and no radiographic studies are indicated.[6,12] If the child fails to meet *all* the criteria for clinical clearance, radiographic imaging is indicated.[6,10]

An initial radiographic examination should include lateral and anterior-posterior views and an open mouth of the cervical spine, which must include the occiput through cervicothoracic junction.[5,12] The open-mouth views are not recommended in children younger than 5 years.[5,14] The use of oblique views and flexion-extension series in pediatric cervical spine trauma is controversial and are recommended only when indicated.[15,16] Oblique radiographs are useful in highlighting detail of facet joints, pedicles, and posterior elements.[12] A flexion-extension radiographic series may be indicated to rule out cervical instability, although only in awake, cooperative, and neurologically intact children with no distracting injury.[3,12]

Further testing is often indicated to further evaluate the cervical spine once abnormalities are identified on plain radiographs or when there is high suspicion despite normal radiographs.[4] Indications for obtaining a CT scan of the cervical spine include bony abnormalities, such as osseous fractures, facet dislocations, compression fractures, or vertebral endplate fractures.[5,12] MRI is best used to evaluate neurologic deficit attributed to spinal cord lesions, soft tissue injuries, posterior ligamentous injury, herniated disk, or edema.[2,3,8] It has also been shown to be effective to rule out occult injuries and in the evaluation of the obtunded patients.[13]

OCCIPITOATLANTAL DISLOCATION

Occipitoatlantal (OA) (Fig. 50-2) dislocation is a relatively rare injury that is more commonly found in children and usually occurs as a result of MVAs.[17] The mechanism has not yet been established. Prompt recognition and early, aggressive surgical management increases the survival rate in the pediatric population.[18]

Most children with OA dislocations present dead on arrival, but if they survive, they can present with a range of clinical neurologic findings depending on the severity of brainstem and cord compression.[19] Some may have incomplete lesions with cranial nerve dysfunctions and varying degrees of quadriplegia, while others may have complete loss of neurologic function below the brainstem. In severe cases, plain radiographic displacement is obvious; however, radiographic assessment can sometimes appear normal. In the past, a Powers ratio greater than 1 was diagnostic for OA dislocations. This ratio is determined by measuring the distance from the posterior arch of the atlas to the basion and dividing this by the distance from the anterior arch of the atlas to the opisthion. However, the most sensitive diagnostic tool according to Harris et al.[20] is a basion-dens interval greater than 12.5 mm, which is the distance between the tip of the dens and the basion. CT also can be used to assess osseous alignment.

Nonoperative measures are inadequate in achieving short- or long-term stability. Therefore, treatment consists of immediate halo immobilization and urgent stabilization and posterior fusion from the occiput to C1 or C2 to preserve remaining neurologic function.[21] Traditionally, internal fixation has been
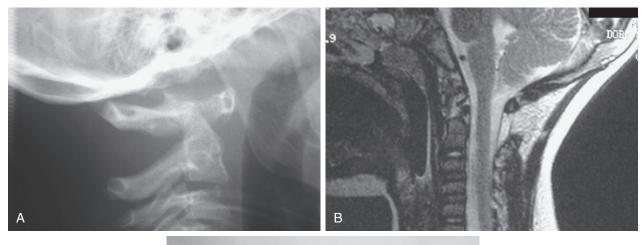

Fig. 50-2 These radiographs are of a 3-year-old boy after a motor vehicle accident in which the patient was an unrestrained passenger thrown from the vehicle. The patient initially had an 8-pin halo applied for closed reduction and was taken to the operating room 2 days later for internal fixation. *A,* The lateral radiograph of the upper cervical spine demonstrates significant abnormalities consistent with occipitoatlantal dislocation and atlantoaxial dissociation. There is increase in the joint space between the occipital condyles and the lateral masses of C1. *B,* The magnetic resonance imaging scan sagittal view shows a distance between the tip of the clivus and the top of the odontoid process of 1.7 cm and an atlantodental distance of 6 mm. There is associated ligamentous injury at C1 and C2. *C,* The lateral radiograph demonstrates posterior fusion of the occiput to C2 with cerclage wires and posterior bone graft at 5 months postoperative.

done with the use of sutures or metal wires placed around the posterior elements of C1 and C2 and through the base of the skull. Rib graft is recommended because the natural curve of the graft provides a close fit to the cervical spine.[21] Postoperatively, the patient can be immobilized by either a halo vest jacket or a halo cast for at least 3 to 4 months, depending on the stability of the fixation and when osseous union occurs.[22] Recently, Schultz et al.[23] has shown that rigid internal fixation produces the same outcome while decreasing the need for rigid external orthotics.

ATLANTOAXIAL ROTATORY FIXATION

Atlantoaxial rotatory fixation (AARF) is a rare disorder that occurs more commonly in children, where the atlas becomes fixed in a position normally achieved during rotation. The etiology is unknown, but thought to be related to inflammation or trauma. A meniscus-like synovial fold within the atlantoaxial joint in children, which undergoes enfolding or rupture as a result of inflammation or effusion from trauma is present in the development of AARF.[24]

Patients with AARF can present with neck pain, headache, loss of cervical motion, and torticollis. Fielding and Hawkins[25] described that most patients with AARF presented with their head tilted and rotated to the opposite side. Patients may also present with facial asymmetry, but neurologic findings are rare. Diagnosis must be confirmed radiographically. A plain anteroposterior, open-mouth radiograph should be taken with the head in the most neutral position possible. In this condition, the radiograph should show a forward-positioned lateral mass that appears wider and closer to the midline, while the other lateral mass appears smaller and farther away from the midline, creating an asymmetrical distance between the dens and the lateral mass. CT evaluation can also aid the diagnosis. To avoid false-positive diagnoses in a patient that has malrotation in a neutral position with guarding from soft tissue injury, Kowalski et al.[26] recommend that the patient be scanned through C1-C2 when he or she initially presents with the heads fixed in lateral rotation, and subsequent scans should be obtained with the head turned to maximum contralateral rotation. These scans must be taken orthogonal to the vertebral bodies to allow accurate visualization. Patients with AARF will show no motion at C1-C2 during this maneuver, whereas patients without AARF will show either a reduction or a reversal of the C1-C2 rotation.

Fielding and Hawkins divided AARF into four types depending on the status of the atlantoaxial ligament. Type I is the most common and benign condition, characterized by unilateral facet subluxation, an intact transverse ligament, and without anterior atlas displacement. Type II occurs with unilateral facet dislocation and displacement of the atlas between 3 mm to 5 mm as a result of an incompetent transverse ligament. In type III, there is bilateral facet dislocation with the atlas being displaced by more than 5 mm resulting from deficiencies in both the transverse and secondary ligaments. Type III is the most serious because all the ligaments are ruptured. Type IV occurs with posterior displacement of the atlas resulting from a deficiency in the dens.

The key to resolving AARF is early diagnosis and treatment.[27] In the acute (<1 week) or subacute (1 to 3 months) setting, regardless of classification, Pang et al. recommend use of halter traction, followed by cervicothoracic immobilization for at least 3 months. Chronic (>3 months) AARF, regardless of classification, are more prone to malalignment than acute or subacute AARF as a result of chronic changes in the ligaments and joint structures.[27] Traction and halo immobilization are recommended for chronic type I patients, which have the highest malalignment rate and worst prognosis for closed reduction. Chronic type II patients should be initially reduced with halter traction and halo immobilization because bracing is usually insufficient. Interestingly, chronic type II patients are more prone to recurrent subluxations and dislocations than chronic type III. Chronic type III patients are managed as in the acute or subacute setting, but if orthosis fails to achieve reduction, traction and halo immobilization are recommended. Recurrences of malalignment in class I or II are treated with traction and halo immobilization, whereas class III is treated with repeat bracing. Further recurrences in acute or subacute AARF are treated with traction and halo, whereas chronic AARF will require posterior C1-C2 fusion. Surgical fusion is recommended if halo immobilization fails or in situations of irreducible deformities.[21]

ATLAS FRACTURES

An atlas fracture, or a Jefferson fracture, rarely occurs in children.[28] It is caused by excessive axial loads applied to the head that cause the occipital condyles to compress and fracture the C1 ring. This can rupture the transverse ligament causing C1-C2 instability. Usually this force is dissipated on the lateral masses, fracturing both the anterior and posterior rings of the atlas. However, in children, this load can result in an isolated ring fracture at the synchondrosis.[29]

These fractures are usually not associated with neurologic deficits. Plain radiographs usually cannot detect atlas fractures, which is why a CT scan is the modality of choice in diagnosing and identifying transverse ligament ruptures. If the transverse ligament is ruptured, flexion-extension radiographs are recommended to evaluate the degree of atlantoaxial instability.

Treatment involves immobilization with the use of rigid bracing such as a Minerva jacket until the fracture heals, which can sometimes take up to 6 months.[30,31] If the transverse ligament is ruptured or there is excessive widening (>7 mm), traction followed by cervical fusion and halo application is recommended.[32]

DENS FRACTURE

Dens fractures (Fig. 50-3) are the most common cervical spine fractures in children younger than 11 years.[33] Most often they are Salter-Harris type I fractures. The mechanism usually is severe trauma from events such as MVAs[34] or falls from significant heights,[35,36] resulting in a fracture of the synchondrosis at the base of the dens. The force usually causes anterior displacement of the dens with posterior angulation.

Odent et al.[34] reported neurologic injuries associated with complete lesions at the level of the cervicothoracic junction in 8 of 15 pediatric patients with dens fractures. This was thought to be due to the anterior displacement of the upper spine, causing spinal cord stretch at the apex of the cervicothoracic spine. Other associated symptoms include persistent neck pain and upper cervical spine tenderness. This injury is also often associated with facial trauma. Diagnosis is made with lateral radiographs that demonstrate anterior displacement of the odontoid. However, in approximately 10% to 15% of cases, the displacement is either posterior or not evident. In this case, flexion/extension radiographs or a CT scan with sagittal images is indicated,[22] which should demonstrate displacement of the odontoid.

Similar to most fractures involving the physis, healing is rapid if the fracture is sufficiently reduced and immobilized. Reduction can be achieved with mild extension of the neck. The goal should be to achieve at least 50% apposition because complete reduction is not necessary for successful healing. Immobilization with a Minerva cast is recommended for children under the age of 3 and a halo for older children.[32] A halo should also be used in patients who cannot achieve sufficient reduction with neck extension. Odent et al.[34] reported successful healing of dens fractures in all 11 patients with the use of a Minerva jacket or a halo cast. The three patients in the study treated operatively had at least one complication. The child should be immobilized for 6 to 10 weeks and can be discontinued when flexion-extension lateral radiographs confirm stability and there is presence of bony healing.

HANGMAN'S FRACTURE

Spondylolisthesis of C2, or Hangman's fractures, are rare in children. They are usually caused by cervical extension and axial loading, causing bilateral pars interarticularis fractures. Often, they are associated with anterior subluxation of
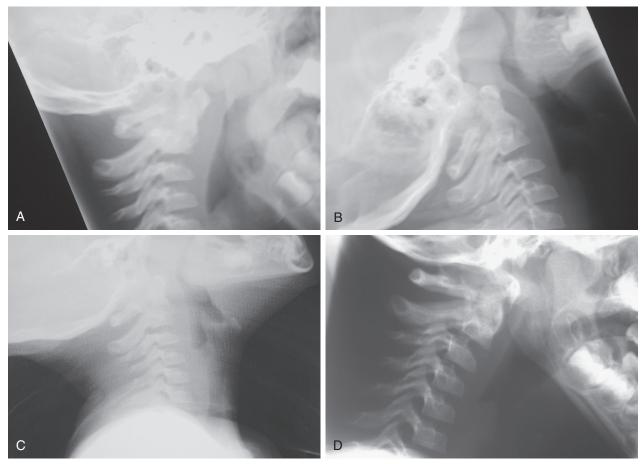

Fig. 50-3 *A,* Lateral radiographs of a 3-year-old boy who sustained a type III dens fracture. *B,* lateral extension radiograph demonstrates closed reduction. *C,* Patient was placed in a Minerva cast for 2 months in an extended position. *D,* Lateral radiograph at 2 months postinjury and casting shows the healed fracture.

C2 and tear or stretching of the anterior longitudinal ligament. These fractures are most likely incurred during MVAs or falls from significant heights. They are more common in children younger than 2 years and is thought that a combination of a relatively large head, poor muscle control, hypermobility of the cervical vertebrae, and child abuse (shaking) contribute to this finding.[37]

Patients often do not present with neurologic deficits. This is due to the ample space surrounding the axial vertebrae and the decompression associated with the injury. However, Pizzutillo et al.[37] reported cases with patients having reported deficits that resolved within a year. This is usually seen in unilateral or bilateral facet dislocations. Diagnosis can be made with lateral and oblique radiographs that demonstrate translucency of the C2 pedicles and often times anterior subluxation of C2 on C3. CT can sometimes aid in the diagnosis, as well as demonstrate the extent of the fracture and the degree of displacement. Care must be taken not to confuse this type of fracture with physiologic anterior displacement.[38] This can be assessed by drawing a line, Swischuk's line, from the anterior aspect of the posterior arch of C1 to the anterior aspect of the posterior arch of C3. If the anterior aspect of the posterior arch of C2 is not within 2 mm of this line, indicating a pseudosubluxation, a fracture can be suspected.[39] However, it should be noted, that sometimes Hangman's fractures can still occur without significant anterior displacement. Smith et al.[40] described additional difficulty with locating the fracture site with the use of CT. The anterior location of the C2 superior articular processes relative to the inferior articular processes causes the pars interarticularis of C2 to be more anteriorly located than any other vertebrae. This causes a Hangman's fracture to sometimes be mistaken for a neurocentral synchondrosis defect leading to a false diagnosis of persistent synchondrosis of the axis on CT.

Treatment depends on the degree of displacement. For anterior displacements less than 3 mm, external immobilization is recommended with either a Minerva or a halo jacket for 2 to 3 months. If the displacement is greater than 3 mm, gentle reduction should be performed to reduce the displacement and the fracture should be immobilized with a halo cast for the same amount of time.[22] If union does not occur and the displacement remains excessive, an anterior cervical fusion involving C2 and C3 is suggested. Pizzutillo et al.[37] reported successful union with external immobilization in four of five patients.

TRAUMATIC ATLANTOAXIAL SUBLUXATION

Traumatic atlantoaxial subluxation (Fig. 50-4) is considered one of the most common types of upper cervical spine dislocations in children.[41] However, McGrory et al.[33] reported that this type of injury is relatively rare and comprises less than 10% of pediatric cervical spine injuries. Atlantoaxial instability or subluxation is often due to traumatic disruption of the transverse ligament of the atlas, which functions to keep the odontoid in contact with the anterior arch of the atlas. De Beer et al.[41] reported this injury occurring with MVAs and sports injuries. In younger children, this also may be due to injury of the neurocentral synchondrosis at the odontoid base.

Patients can present with neck pain and/or neurologic deficits. Approximately one third of the anterior portion of the canal consists of the odontoid, the middle third is occupied by the cord, and the remaining third is unoccupied area, which normally allows for less than 4.5 mm of physiologic C1-C2 translation.[42,43] When this exceeds 5 mm, as a result of disruption of the transverse ligament, the spinal cord can

Fig. 50-4 These radiographs are of a 4-year-old boy after a motor vehicle accident in which he sustained a C1-C2 subluxation. *A,* The patient was placed in a halo for 3 months, at which time he still demonstrated 8 mm atlanto-dens interval with minimal forward flexion. The patient was then scheduled for surgery for persistent instability. *b,* Four years out, the lateral radiograph demonstrates solid fusion of C1-C2 via sublaminar wires. Incidentally, add-on fusion of C3 took place.

be impinged resulting in neurologic deficits. Evaluation is done by assessing the atlanto-dens interval (ADI), which is the distance between the anterior cortex of the odontoid and the posterior cortex of the arch of C1, on both lateral radiographs and flexion-extension views. A diagnosis can be made if the distance exceeds 5 mm on flexion-extension views. If neurologic symptoms are present, an MRI can be used to evaluate the degree of cord impingement.

In adults, cervical fusion is indicated to prevent neurologic injury. However, in children, the indications are less well defined. De Beer et al.[41] described conservative immobilizing treatment of bracing with extension with an orthosis or a Minerva cast for 8 weeks. Three out of the four patients remained stable neurologically, but some of these patients had increases in the ADI and the fourth patient incurred a serious neurologic injury requiring arthrodesis. For asymptomatic, neurologically intact patients with 5 mm to 8 mm of cervical instability, external immobilization with the use of a Minerva cast or an orthosis for at least 2 months should allow sufficient time for the ligament to heal. For asymptomatic patients whose cervical instability is more than 8 mm or for patients with 5 mm to 8 mm of cervical instability with evidence of spinal cord impingement or damage, cervical arthrodesis is indicated. Follow-up flexion-extension radiographs should be done to evaluate the degree of remaining instability after treatment and may possibly require further treatment.

SUBAXIAL SPINE INJURIES

Fractures and dislocations of the subaxial (C3 to C7) spine are relatively rare in younger children but occur more often in older children and adolescents and have comparable incidence rates to adults. McGrory et al.[33] reported that 67 of 143 (47%) cervical spine fractures take place between C4 and C7 and predominantly occur in 11 to 15 year olds. Some of the more common subaxial injuries are compression fractures, facet dislocations, fracture with dislocation, burst fractures, and posterior ligament injuries. Treatment in older children and adolescents follow adult patterns and therefore standard treatment is indicated.

Compression fractures are relatively rare among the subaxial spinal injuries. McGrory et al.[44] reported that 10 of the 67 (15%) subaxial spine fractures were compression fractures. They are due to hyperflexion without rotation of the cervical spine leading to wedging of the anterior portion of the vertebral body. The posterior ligamentous structures and the posterior aspect of the vertebral body remain intact, and there is no bone or disk protrusion into the spinal canal. As a result, neurologic injuries are rare. Diagnosis is made by lateral radiographs that demonstrate anterior wedging; however, in children the normal anterior wedge shape of the vertebral body can make diagnosis difficult. Surgical treatment is usually not necessary, but cervical spine immobilization in a cervical collar for 2 to 4 months is recommended.

The duration can be longer depending on the extent of the injury and the age of the child.[22]

Facet dislocations can either be unilateral or bilateral, and can be complicated by subluxation (Fig. 50-5). McGrory et al.[33] reported one patient each (1%) with unilateral and bilateral facet dislocations and 11 patients (16%) with facet fractures and subluxations. These injuries are usually caused by hyperflexion with rotation of the cervical spine. Patients can present with neurologic findings as a result of nerve root and/or spinal cord compression. Bilateral facet dislocations are more prone than unilateral dislocations to neurologic injury as a result of the increased instability. Diagnosis can be made with lateral radiographs that demonstrate facet subluxations and an MRI can demonstrate the extent of cord involvement. It is, however, controversial whether a prereduction MRI is needed to detect unrecognized cervical disk herniations. Hart et al.[45] argue that prereduction MRIs are unnecessary and delay treatment in unilateral cervical facet dislocations. Treatment involves gradual distraction and reduction maneuvers by slightly flexing the neck followed by extension. The patient should then be immobilized in a halo cast for 2 to 3 months. Alignment and reduction should be confirmed with radiographs. If reduction is not achieved, an open reduction with primary fusion and internal fixation with posterior wiring is indicated. The patient should then be immobilized in a halo cast for 2 to 4 months.[22]

Fracture with dislocation of the subaxial spine is among the most common subaxial injuries as reported by Birney et al.[46] It is typically caused by trauma and is seen in events such as MVA or falls with direct blows to the head. Patients

Fig. 50-5 Lateral radiograph of a C2-C3 unilateral facet dislocation sustained by a 7-year-old girl after a fall off a swing. She presented with a right-facing cock-robin deformity. She reported numbness of her left ear and burning and stinging on the posterior aspect of her neck. The patient was placed in a SOMI cervical thoracic orthosis in which the deformity progressed over 1 week. She was then taken to the operating room for open reduction and fusion.

can present with neurologic deficits when the cord is compressed secondary to dislocation. Diagnosis can be aided with the use of plain radiographs that demonstrate fracture and dislocation, and an MRI is used to evaluate the extent of spinal cord injury. Treatment involves reduction with external immobilization.

Burst fractures are relatively rare among the subaxial injuries. McGrory et al.[33] reported a total of six patients (9%) with burst fractures. They are caused by excessive axial loads applied to the head in a slightly flexed position. This is commonly seen in football injuries. The typical burst fracture is called a teardrop fracture, whereby there is anterior displacement of the anteroinferior aspect of the vertebral body and a sagittal fracture of the posterior aspect of the vertebral body with protrusion into the canal.[22] As a result, patients often present with signs of cord compression. Diagnosis can be made with plain radiographs; however, the canal should be assessed by CT, and MRI should be used to evaluate the extent of posterior ligament injury and disk herniation. A nonsurgical approach should be the first choice. In patients with neurologic injury, gentle closed reduction with a halo-cast immobilization for 2 to 3 months should be performed.[22] If neurologic deficits are present and spinal cord compression persists despite the prior procedure, an anterior decompression with removal of retropulsed fragments and use of strut grafting is indicated.[22] However, if there is significant posterior ligamentous instability, posterior stabilization should be added to the treatment regimen.

The last type of injury is damage to the posterior spinal ligaments. Most of these injuries occur in children older than 10 years.[22] This is usually caused by a flexion-rotation mechanism that leads to tearing of the posterior ligaments and the facet joint capsules. When a significant amount of force is applied in a flexion-rotation manner, the posterior ligamentous injuries can be complicated by unilateral or bilateral perched facets. In the case of a pure ligamentous injury, patients are neurologically intact; however, ligamentous injury accompanied by perched facets can result in neurologic deficits. Diagnosis can be aided by the use of radiographs that demonstrate instability. Because most of these injuries occur in children over the age of 10, an adult criterion is used to assess the degree of instability. Instability is defined as either angulation between adjacent vertebrae in the sagittal plane of greater than 11 degrees or translation in the sagittal plane of 3.5 mm or more.[22] Treatment for minor instability is external immobilization; however, injury with significant instability requires posterior fusion.[22]

SPINAL CORD INJURY WITHOUT RADIOGRAPHIC ABNORMALITY

Spinal cord injury without radiographic abnormality (SCIWORA) is a term used to describe an acute spinal cord injury that may result in neurologic deficits without evidence of vertebral fractures or malalignment on plain radiographs

and CT scans. These injuries are relatively common (19% to 34% of all spinal cord injuries)[47] and more commonly occur in pediatric populations. This is thought to be due to the fact that children have more horizontal facets allowing for increased translational motion in the anteroposterior plane, anterior wedging of the vertebral bodies, and ligaments and joint capsules with greater elasticity as compared with adults.[47,48] These types of injuries most commonly occur in MVAs, falls from significant heights, and sports injuries. Patients can present with a wide variety of sensory and motor deficits depending on the level and extent of spinal cord and nerve root injury. Diagnosis is made from a neurologic examination with focal deficits and negative radiographic and CT findings and can be aided with MRI scans that demonstrate the extent of neural injury, as well as associated hemorrhage and edema. Treatment involves the use of corticosteroid and external immobilization for at least 3 months.[47,48] Surgery is indicated only in patients with spinal instability or cord compression secondary to extraneural injury.[47]

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Evaluation and Surgical Management of Thoracic/Thoracolumbar Junction Injuries in Children

INTRODUCTION

Fractures of the thoracolumbar (TL) spine in children are uncommon injuries, accounting for less than 5% of all acute spinal injuries.[1] They often occur after high-energy trauma such as motor vehicle accidents, and other injuries should always be considered. The levels, patterns, and mechanisms of injury to the skeletally immature axial skeleton are influenced by the different anatomic and biomechanical properties of the developing spine. These traumatic injuries can be quite different in children than adults, and the often quoted "Children are not small adults," is never more true. Treatment considerations must take into account stability of the spine fractures, the neurologic status of the patient, and the dynamic elements of growth and remodeling. Vertebral body height can be restored after compression fractures with subsequent growth secondary to remodeling.[2] On the other hand, further growth of the spinal column in a child with a spinal cord injury may contribute to a progressive spinal deformity such as scoliosis.[3–5]

EPIDEMIOLOGY

The true incidence of fractures and injuries involving the thoracic and lumbar spine in children is not well defined. The majority of studies tend to include cervical spine

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injuries in the analysis of pediatric spine fractures. Despite this limitation, the literature supports the concept that the majority of TL fractures occur in the second decade of life, and thoracic fractures (T4 to T12) are most common, followed by fractures at the TL junction (T12 to L2).[6–8] Fractures at multiple levels, resulting from the increased flexibility of the spine and hydration of the intervertebral disks, have been reported to occur in 35% to 40% of patients.[1,7,9] In older children and adolescents, the most common cause of injury occurs after motor vehicle accidents, followed by falls and recreational/sports injuries.[1,6,7] An associated neurologic injury is present in 26% of patients.[1,10] The incidence of pediatric spine injuries peaks in two age groups: children 5 years of age or younger, and children 11 years of age or older.[11,12]

MECHANISMS OF INJURY/ PATTERNS OF INJURY

Different mechanisms of injury predispose children to spinal trauma depending on their age at the time of injury. Infants and toddlers are prone to spinal trauma after falls and motor vehicle accidents. Adolescents sustain traumatic spinal injuries following sports and recreational activities such as diving.[13] The three-column classification scheme used in adults (Denis) has not been commonly applied to pediatric or adolescent patients.[14] No one has yet reviewed a series of pediatric patients to apply the three-column classification system. These injuries are most often described as being a result of compression, distraction, or shear.

Compression fractures occur primarily in the thoracic spine resulting from flexion injuries. These fractures tend to be stable, involving only the anterior column of the spine. The thoracic rib cage provides inherent stability to these injuries. Wedging of the anterior vertebral bodies with less than 50% loss of anterior vertebral height is often noted. Multiple contiguous compression fractures can be found, resulting from the increased flexibility of the spine and hydration of the intervertebral disks, which enables the forces to be dissipated over several levels.[15] The increased elasticity of the posterior ligamentous complex is thought to contribute to the increased incidence of multiple compression fractures

noted at the TL junction and lumbar spine following lap-belt injuries.[16]

Burst fractures comprise only about 10% of all pediatric TL fractures and are more common in older children approaching skeletal maturity. The more mobile and elastic skeletally immature spinal column dissipates compressive forces that would typically result in burst fractures in adults.[17,18] Burst fractures result from axial load and flexion forces applied to the spine. Failure of the middle column occurs as the vertebral endplate fails and the intervertebral disk is forced into the vertebral body leading to retropulsion of bone into the spinal canal (Fig. 51-1). The most common level of injury is the TL junction. Compared with adults, the spinal canal is larger relative to the spinal cord in children, and a greater degree of canal compromise is tolerated without impingement of the cord or cauda equina. Burst fractures occurring distal to the L1 level tend to also have a lower incidence of permanent neurologic injury, as the cauda equina is more tolerant of compression than the spinal cord. Multiple-level TL burst fractures have recently been reported. The proximal fracture level is most often responsible for the neurologic deficit when present.[19]

The mandatory use of seat belt restraint systems has increased the incidence of flexion-distraction or Chance fractures of the lumbar spine in children. Four types of flexion-distraction injuries have been described which are defined according to the distraction plane of injury through the ligaments, bone, or the intervertebral endplates[20] (Fig. 51-2). The classic Chance fracture is a bony injury through the vertebral body, the pedicles, and the spinous processes.[21] A

Fig. 51-1 CT scan demonstrating failure of the middle column with retropulsion of bone fragments into the spinal canal.

ligamentous Chance fracture exits through the disk space anteriorly. Other variations include pure facet dislocation with disruption of the disk space and dislocation through the facet joints with fracture through the body. The unique characteristics of the child's small pelvis, higher center of gravity, and their typical slouched posture prevents the normal or intended positioning of the lap belt across the pelvis.[22,23] At the time of frontal impact, the properly positioned lap belt is intended to dissipate the deceleration forces through the pelvis and the hips. Children tend to submarine under the seat belt and the lap belt improperly rides up to the level of the mid-lumbar spine level. The lap belt then acts as an anterior fulcrum that generates the flexion-distraction mechanism of injury (Fig. 51-3). In addition to spinal column injury, there may be associated injury to the spinal cord resulting in paraplegia and life-threatening visceral injury.[20,23–27]

Fractures involving the vertebral apophysis are unique to the developing spine and are often reported as isolated case reports.[28,29] The ring apophysis contributes to the developing contour and width of the vertebral body during growth. The strength of the intervertebral disk and its attachments in children allows excessive forces to be transmitted to the relatively weak cartilaginous vertebral endplate. The actual fracture is through the hypertrophic zone of the growth plate, and the inferior endplate is more commonly involved. Under a significant single or repetitive axial load, the apophysis can slip or separate into the spinal canal and mimic the clinical findings of a herniated intervertebral disk. Mechanisms of injury have included weight lifting, gymnastics, hyperextension, and trauma. These patients present with back and leg pain, often without significant neurologic signs.

Several reports in the literature have increased our awareness that spine trauma can occur as part of the spectrum of injuries in the battered child syndrome. The true incidence of spinal injuries as the direct result of abuse is not known but is estimated to be about 3%.[30] In one series, the average age of a child with a spine fracture resulting from child abuse was 22 months old.[31] Most of the spinal fractures resulting from child abuse involve the vertebral body in the region of the TL or lumbar spine. Many of these spine injuries are not recognized initially because of the lack of neurologic involvement and the absence of gross deformity. The majority of compression fractures secondary to abuse are usually mild (less than 25% wedging).[31] Child abuse or pathologic etiologies must be considered when TL fractures are seen in children. TL fractures with listhesis have been reported as a manifestation of nonaccidental trauma in seven children ages 6 months to 7 years. Fracture patterns varied from subtle listhesis to frank vertebral dislocation, and two children became paraplegic. Paravertebral calcification was present in all but one case and in two children the presence of TL fracture was the only radiographic sign of abuse.[32] The presence of multiple spinous process fractures or multiple compression fractures on radiographs should raise one's index of suspicion
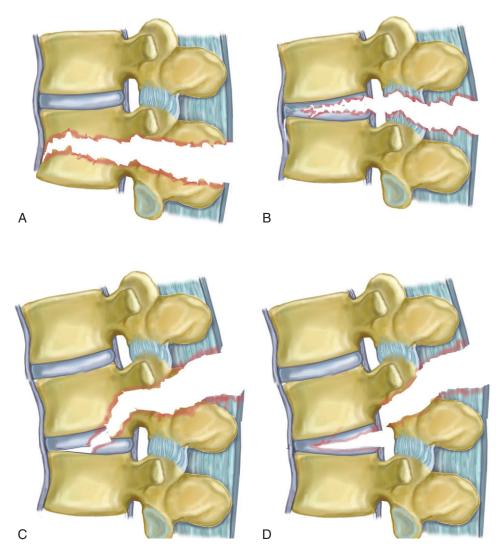
A

B

C

D

Fig. 51-2 Flexion-distraction classification defining distraction plane of injury through ligaments, bone, or intervertebral endplates. (From Rumball K, Jarvis J: Seat belt injuries of the spine in young children. J Bone Joint Surg 74B:571–574, 1992.)

Fig. 51-3 Lap belt acts as an anterior fulcrum that generates the flexion-distraction mechanism of injury. (From Johnson DL, Falci S: The diagnosis and treatment of pediatric lumbar spine injuries caused by rear seat lap belts. Neurosurgery 26:434–441, 1990.)

for child abuse. In suspected cases, a lateral radiograph of the TL spine should be part of the routine skeletal survey.

CLINICAL EVALUATION

The majority of acute TL fractures result from relatively high-energy trauma. Concomitant fractures are common due to the significant force required to produce these fractures. Infants or children with suspected spine injuries should be properly immobilized to prevent motion that could produce further injury. Careful log rolling and other spinal precautions should be maintained until appropriate screening radiographs can be obtained.

The clinical examination of a child with a suspected spine injury can be extremely difficult. Often the child is quite young and frightened and unable to communicate the location of the pain. The physical examination must closely evaluate for tenderness along the TL spine. Evaluation of the

skin for any cutaneous signs such as abrasions or bruising is important. The presence of a bandlike pattern of ecchymosis about the abdomen or iliac crest is described as a "lap belt" sign and may indicate an underlying lumbar spine fracture and associated visceral injuries (Fig. 51-4). A thorough neurologic examination should be performed to evaluate motor strength, sensation, reflexes, and proprioception. A rectal examination to evaluate for the bulbocavernosus reflex should be done in children with suspected acute spinal cord injury to determine when the period of spinal shock is over.

CLINICAL MANAGEMENT

Imaging of the pediatric spine should provide full assessment of the specific level(s) of injury, the relative stability of the fracture, and the pattern and cause of spinal cord injury if present. Standard anteroposterior (AP) and lateral radiographs of the TL spine will often demonstrate the type of injury in the older child and adolescent and the overall alignment and/or displacement of the spine. After a spine fracture has been identified, computed tomography (CT) scans are essential to assess the extent of osseous injury. This includes the integrity of the middle column (determining the degree of canal compromise in burst fractures) and of the posterior column (an unrecognized lamina or facet fracture). Three-dimensional reconstruction CT can help in evaluating areas difficult to see on plain radiographs such as the cervicothoracic junction and the thoracic spine. Measurements used to assess fracture stability that may influence treatment decisions in adults (degree of kyphosis, loss of anterior vertebral height, and the degree of canal compromise) have not been well defined in pediatric spine fractures, and each fracture should be treated on an individual basis.

Magnetic resonance imaging (MRI) should be obtained in children presenting with a spine fracture and an associated neurologic injury. The increased elasticity of the ligamentous complex of the spinal column in response to high-energy trauma may result in traumatic spinal cord injury without radiographic abnormality (SCIWORA), and MRI is also warranted in the child with suspected SCIWORA to evaluate the location and extent of spinal cord injury.[5,33,34] This may occur when a force applied to the flexible spine exceeds the tensile limits of the relatively inelastic spinal cord, and result in a distraction or an ischemic injury to the spinal cord. Thoracic SCIWORA is an important subset of this unique injury pattern. Three subtypes/causes have been identified: (1) after high-speed direct impact, (2) distraction from lap belts, and (3) crush injury by slow-moving vehicles.[34] Advancements in the techniques of MRI have refined the evaluation of injuries to the spinal cord, ligaments, and other soft tissues, the intervertebral disks, and the cartilaginous or unossified pediatric spine (Fig. 51-5). Slipped vertebral apophyseal injuries are unique pediatric spine injuries readily identified by MRI. Three distinct patterns of injury to the spinal cord has been noted on T2-weighted images.[1,35] One recent retrospective study concluded that MRI is the imaging modality of choice because it could accurately classify injuries to the bones and ligaments, and spinal cord patterns determined by MRI have predictive value.[35]

NONOPERATIVE APPROACHES

Nonoperative management of pediatric TL fractures requires consideration of the neurologic status of the patient, the stability of the fracture pattern, coronal and sagittal plane

Fig. 51-4 Lap-belt sign: Bandlike pattern of ecchymosis about the abdomen or iliac crest may indicate an underlying lumbar spine fracture and associated visceral injuries.

Fig. 51-5 MRI demonstrating thoracic burst fracture at two contiguous levels (T5-T6) with cord compression and incomplete lesion.

deformities, and other associated injuries. In general, the majority of these injuries are stable, the risk of spinal cord injury is lower compared with adults, and most can be treated conservatively. Fractures involving the spinous or transverse processes can be treated on a symptomatic basis. They often result from high-energy trauma, and their identification requires observation for potential intra-abdominal injuries.

The majority of compression fractures (often with <50% loss anterior vertebral height) can be managed conservatively, often with an extension brace for 6 to 8 weeks. Patients with multiple compression fractures should be followed for longer periods to monitor for a progressive kyphotic deformity. Remodeling of compression fractures occurs more often in younger children. Flexion-distraction or Chance fractures, in which the fracture line passes primarily through bone, will heal with an 8- to 12-week course extension cast and brace immobilization. Pantaloon casts including one extremity increase the stability of the fractures involving the lumbar spine. It is important to maintain a high index of suspicion for associated intra-abdominal or retroperitoneal injuries with flexion-distraction injuries and consider delaying cast application until these have been ruled out.

Burst fractures that are thought to be stable, after taking into consideration the patient's age, associated injuries, and the degree of deformity, can be treated nonoperatively. Burst fractures occurring distal to the L1 level tend to have a lower incidence of permanent neurologic injury because the cauda equina is more tolerant of compression than the spinal cord. Treatment consists of a 4- to 6-week period of bed rest, followed by hyperextension casting or bracing for an additional 8 to 12 weeks. The degree of canal compromise measured on CT scan is usually not a factor in the treatment decision-making process of these fractures. In addition to the larger relative canal dimensions in children, resorption of retropulsed bone fragments predictably occurs with remodeling.[36] Similar to patients with multiple compression fractures, these patients must be followed for longer periods of time to observe for progressive kyphotic deformities.

INDICATIONS AND CONTRAINDICATIONS FOR SURGERY

Disruption of the posterior ligamentous complex usually results in an unstable fracture pattern that will not heal on its own. Surgery is generally indicated for patients with unstable fractures, progressive kyphotic deformity, or with neurologic injury.[4] Unstable burst fractures result from combined compressive failure of the anterior and middle columns and tensile failure of the posterior elements. Proposed radiographic indicators of potential instability applied to adults include more than 20-degree kyphosis, more than 50% loss of vertebral body height, and more than 40% canal compromise. However, these criteria have not traditionally been applied to

the skeletally immature child but may apply to the skeletally mature adolescent approaching the end of growth. If there is no neurologic deficit, the degree of spinal canal compromise associated with a burst fracture is usually not a factor in decision making for treatment of these fractures. Operative decompression and stabilization with internal fixation is indicated for unstable burst fractures in patients with neurologic deficits (Fig. 51-6, *A* to *C*). True Chance fractures entirely through the bone have been shown to heal quite predictably. Soft tissue Chance fractures can occasionally be treated nonoperatively. A periosteal sleeve fracture, often interpreted as a lumbar dislocation in young children, can heal well and result in long-term spinal stability.[37] The more common purely ligamentous flexion distraction injuries are treated surgically and are stabilized by restoring the posterior tension band. Operative stabilization of a flexion distraction injury may be indicated when it is associated with an abdominal injury. Hyperextension casting may not be permissible and internal fixation of the fracture would promote early mobilization.

Children with incomplete spinal cord injuries should be treated with expectant waiting for neurologic improvement. For those children with a complete spinal cord injury, the incidence of progressive spinal deformity approaches 100%.[3] It is related to the age of the child, the degree of spasticity, and the level of the spinal cord injury. Treatment should be initiated for anticipated progressive spinal deformity before it becomes severe.

OPERATIVE PROCEDURE AND TECHNIQUE

The primary goals in the operative treatment of pediatric spine fractures are directed toward positively influencing the natural history of the injury. These would include decompression of spinal fractures to maximize neurologic recovery, restoring normal coronal and sagittal plane alignment, and preventing progressive deformities. Fracture-dislocations and "unstable" burst fractures usually require operative intervention. These often can be treated with a posterior approach using indirect reduction techniques that rely on an intact posterior longitudinal ligament. Preserving motion segments or spinal mobility by limiting the extent of posterior segmental instrumentation should also be considered, particularly in the lower lumbar spine. One exception to this concept involves patients with complete spinal cord injuries, who require longer instrumentation and fusion constructs to prevent progressive deformity.[3,5] Patients with an incomplete spinal cord injury may benefit from a direct anterior approach and reduction. These most often occur in older children and adolescents approaching skeletal maturity and can be treated with anterior approaches and instrumentation similar to adult patients.

The most common operative indication for spine fractures in children is true ligamentous flexion-distraction injuries. These are addressed from a posterior approach to the spine.
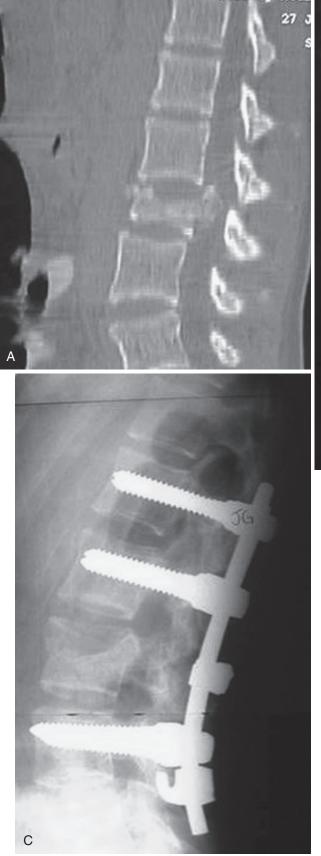
Fig. 51-6 *A,* CT lateral reconstruction demonstrating an L3 burst fracture in a 14-year-old child following a motor vehicle accident, neurologically intact. *B,* MRI of L3 burst fracture. *C,* Postoperative lateral radiograph following L1-L4 posterior spine fusion and instrumentation with indirect decompression of L3 burst fracture.

Associated retroperitoneal or other visceral injuries must be ruled out and treated before stabilizing the spine injury. After the child is intubated, electrodes are placed for spinal cord monitoring prior to turning to the prone position. At our institution, trans-cranial somatosensory evoked potential (SSEP) and motor evoked potential (MEP) are obtained, and level-specific electromyographic (EMG) monitoring is used if pedicle screws are planned. The patient is then carefully turned to the prone position after baseline potentials are obtained, and positioned on chest rolls on a radiolucent operating room (OR) table. The involved levels are identified by c-arm fluoroscopy to limit exposure of the posterior spine. It is not uncommon to see disruption of the lumbo-dorsal fascia after midline exposure. Care is taken exposing the posterior elements of the spine out to the transverse processes. The underlying dura, spinal cord, or nerve roots may be exposed with disruption of the interspinous ligaments and ligamentum flavum. The extent and degree of injury should be determined using both electrocautery and elevators to minimize the risk of inadver-

tent injury. Reduction of the flexion-distraction injury is achieved with a compression construct limited to the involved levels of the injury. Multiple levels of involvement may be present in pediatric spine fractures and should be considered in operative planning and treatment.[16] The options for internal fixation of the spine depend on the size of the child and surgeon's preference. In young children, spinous process wiring can be used (Fig. 51-7). Compression constructs using hooks or pedicle screws and rods may be used in older children (Fig. 51-8, *A* to *D*). Anatomic reduction, facet ablation, decortication, and bone grafting (allograft or iliac crest) are critical to achieving a solid arthrodesis of the spine. Intraoperative spinal cord monitoring is important during the reduction maneuvers and instrumentation of the spine. Cast immobilization is used for 6 to 12 weeks postoperatively. A pantaloon cast is applied usually a few days after the procedure to monitor for any occult abdominal injuries in the immediate postoperative period. On rare occasions, prominent hardware may be removed if symptomatic after fusion has been achieved.
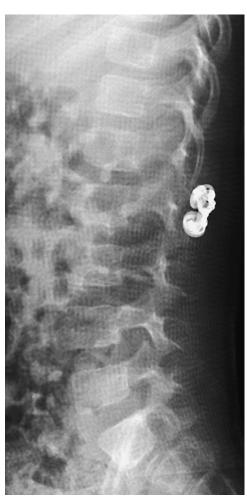

Fig. 51-7 Postoperative lateral radiograph of L1-L2 Chance fracture using wire fixation in a 5-year-old child.
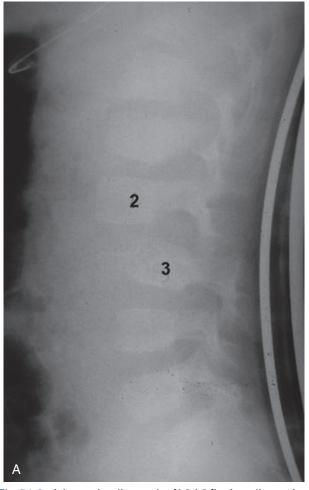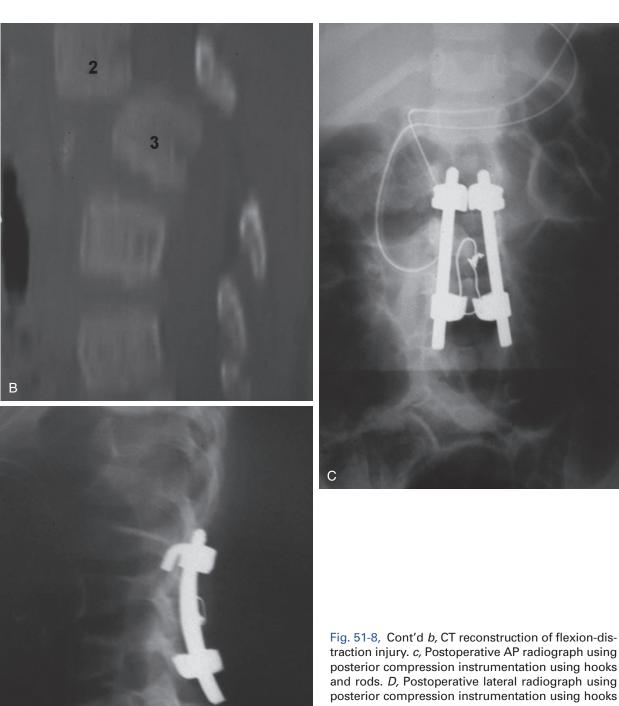

Fig. 51-8 *A,* Lateral radiograph of L2-L3 flexion-distraction injury in a neurologically intact 4-year-old child following lap-belt restraint.

Continued

B

C

D

Fig. 51-8, Cont'd *b,* CT reconstruction of flexion-distraction injury. *c,* Postoperative AP radiograph using posterior compression instrumentation using hooks and rods. *D,* Postoperative lateral radiograph using posterior compression instrumentation using hooks and rods.

CONCLUSION

Fractures of the TL spine are relatively uncommon injuries in children. The patterns of injury are influenced by the anatomic and biomechanical properties of the growing spine. The goals of management include achieving spinal stability, accommodation of the neural elements, and pain-free function.

Fortunately the majority of these injuries can be treated conservatively. Remodeling predictably restores vertebral height following compression fractures, and the spinal canal seems more tolerant of canal compromise resulting in fewer neurologic injuries. The most common injury necessitating operative stabilization in the skeletally immature patient is the ligamentous flexion distraction injury. The goal of surgery is to restore the posterior tension band with a short-segmental construct. Following careful attention to the patient and the pattern of injury in the acute setting, significant long-term post-traumatic deformities and sequelae are rare.

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CHAPTER 52

SUKEN A. SHAH,

KEISHA DEPASS

Evaluation and Surgical Management of Lumbar Injuries in Children

INTRODUCTION

The distribution of injury to the vertebral column varies between the adult and pediatric population because of the age-dependent anatomic differences in the axial skeleton. Each year, approximately 1000 new spinal injuries in children are reported.[1] The cervical spine is the most commonly injured site in this patient population secondary to a larger head-to-body ratio. The incidence of injury in this region has been reported to be as high as 60% of all spinal injuries. Lumbar spine injuries account for approximately 22% of spine injuries in children.[2] Such injuries might result from acute traumatic events or repetitive or overuse syndromes. The incidence of lumbar injuries has continued to fluctuate with major legal and social changes in our society. The change in automobile safety requirements has had a significant impact on the incidence of lumbar flexion-distraction fractures. The increasing popularity in the United States of certain sports (e.g., soccer) has lead to an increased prevalence of spondylolysis in the younger population.[3] It is therefore important for the medical team to be able to diagnose these injuries and manage them accordingly.

In the trauma setting, the medical team often is more focused on cervical immobilization and evaluation. Recognizing specific mechanisms and/or physical findings, such as "the seat belt sign" (discussed in this chapter), will help to direct the team toward further evaluation of the lower spine. In addition, lumbar spine fractures have a higher association with gastrointestinal and other orthopedic injuries than do thoracic and cervical fractures combined.[2]

The injuries can be life threatening and should be recognized quickly at presentation.

Multiple factors contribute to the difficulties of making the diagnosis of pediatric lumbar spine injury. Young children are difficult to examine and often cry on approach, making localization of the level of injury very difficult. In addition, the lack of familiarity with the anatomy of the developing lumbar spine can lead to difficulty with interpretation of commonly used modalities, such as radiography and computed tomography (CT). The presence of multiple ossification centers and the age-related changes that occur in the spine make it difficult for the unseasoned practitioner to differentiate between normal and abnormal. In this chapter, we review the anatomy of the pediatric lumbar spine from birth to adolescence. We also review the different mechanisms of injury, fracture type, and management techniques. For the purpose of this chapter, we will define the lumbar spine as the region from the L2 vertebral body to the L5-S1 disk space. The thoracolumbar region, which has its own unique considerations, is discussed in Chapter 51.

ANATOMY AND IMAGING OF THE PEDIATRIC LUMBAR SPINE

The vertebrae and the spinal canal diameter of the lumbar spine are larger than those of its counterparts in the cervical and thoracic spine. The L3 and/or L4 vertebrae often are the largest when evaluating sagittal diameter.[4] The epidural space varies in content when comparing the upper lumbar with the lower lumbar region. In the upper lumbar spine, little epidural fat is present, but as one approaches S1, the epidural space contains larger deposits of fatty tissue. The diameter of the spinal canal decreases from L1 to L3, with a slight increase at L5 to a size not larger than the initial size at L1. Posterior to each vertebral body is a large network of epidural veins: the retro vertebral plexus. The spinal cord terminates at the conus medullaris, usually at the L1-L2 level, and the remainder of the dural sac is filled with the cauda equina.

At birth, the vertebral bodies are made up mostly of hyaline cartilage. Ossification centers are present, primarily in the central portion of the vertebral body. By 3 months of age, the ossification centers have increased in size and vascular

channels exist throughout the surrounding cartilage. The neural foramina in the lumbar spine seem to be more capacious than those in adults.[5] The nucleus pulposus occupies 75% of the central portion of the disks. Fine linear structures might present within the pulposus that represent notochordal remnants and can be visualized with magnetic resonance imaging (MRI) of the infant spine. The annulus of the disk and the hyaline cartilage of the vertebral bodies have the same intensity on magnetic resonance images and therefore

are difficult to differentiate. Posterior to the vertebral bodies but anterior to the posterior longitudinal ligament is the area that contains the retro vertebral plexus.

By the age of 10 years, ossification of the body is almost complete. The neural arch is also ossified. The nucleus pulposus takes a more eccentric position in the disk and occupies less than 50% of its sagittal diameter. In the upper lumbar spine, this is located in a more posterior position and moves anteriorly at the more caudal disk spaces (Fig. 52-1). Disks undergo
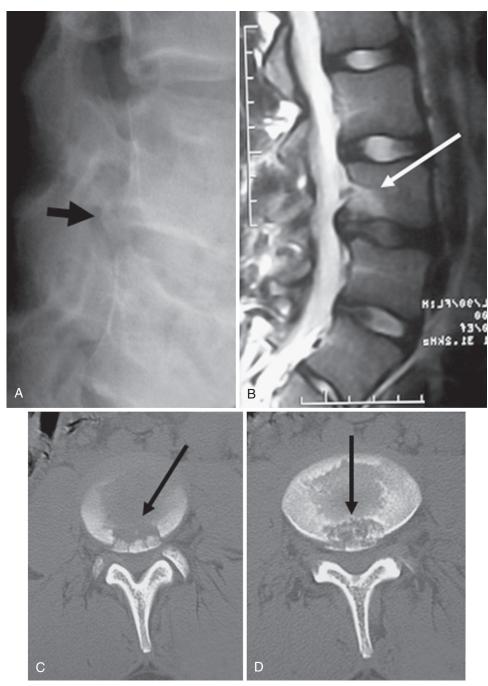

Fig. 52-1 *A,* Magnetic resonance image of the lumbar spine of a 3-month-old patient. H, hyaline cartilage; A, annulus fibrosis; S, Sharpey's fibers; N, nucleus pulposus. B, Magnetic resonance image of the lumbar spine of a 10-year-old patient. F, fibrous tissue. *(Reproduced with permission from Yu S, Haughton V, Rosenbaum A: Magnetic resonance imaging and anatomy of the spine. Radiol Clin North Am 229:691–709, 1991.)*

their own growth and evolution and can be classified as immature, as seen in the newborn, or as transitional, as seen in the adolescent population. The adult, early degenerated, and severely degenerated disks more commonly are seen in persons older than 30 years. MRI findings of disk degeneration have been observed for children as young as 10 years, but these are not necessarily symptomatic.[6] The disk is attached to the vertebral body through Sharpey's fibers of the outer annulus. These fibers are embedded in the ring apophysis of the vertebral body. The ring apophysis does not fuse to the adjacent vertebral body until approximately 18 years of age and is an area of potential weakness. In some studies, this has been linked to lumbar disk herniations[7] and spondylolisthesis.[8]

Pedicular morphology also changes as the child ages. In all ages, the transverse pedicle diameter increases as one moves from L2 to L5. L5 tends to have the shortest pedicles in length and L3 has the longest. These lengths increased directly as the child gets older. The transverse plane angulations of the pedicles, however, have an inverse relationship to age. As the child ages, the angle decreases.[9] The angulation increases as one moves from L2 to L5. In the sagittal plane, an angle between the superior endplate and a line drawn through the midpoint of the pedicle converging at the anterior/superior corner of the vertebral body decreases as one moves from L2 to L5.

Imaging of the immature spine with CT can be difficult because in younger patients, a significant portion of the spine is unossified. In addition, distraction injuries and apophyseal fractures are difficult to exclude with CT. Many now advocate the use of MRI earlier in the process of evaluation because of its sensitivity. In adults, MRI classifications of cord changes have been used to predict outcome. In pediatric patients, these MRI findings have also been shown to have a predictive value for outcome.[10] Most lumbar spine injuries occur below the level of the conus, and currently no MRI classifications predict outcomes with cauda equina injuries.

MECHANISM OF INJURY

The severity of injury is directly related to the mechanism of injury. However, similar mechanisms can cause different injury patterns in different age groups. Motor vehicle accidents (MVAs) by far cause the majority of spinal injuries in children. However, when one divides the pediatric population into groups based on age—infants, toddlers, school age children, and adolescents—differences are apparent. MVAs cause the highest percentage of all types of spinal injuries in infants when compared with other mechanisms, such as falls, sports participation, and other recreational activities. In the toddler and school-age populations, the largest percentage of spinal injuries are caused by falls. In adolescence, 29% of spinal injuries occur secondary to sports participation. Lumbar spine injuries rarely occur during infancy and occur more often in the toddler, school age, and adolescent populations.

The seat belt syndrome was initially described by Garrett and Braunstein[11] in 1962 as a constellation of signs and physical findings in children who are restrained back seat passengers

with an isolated lap seat belt involved in head-on collisions. In the early 1980s, the majority of backseat passengers involved in significant collisions died as a result of their injuries. It was not until the Transportation Safety Board made rear lap belts mandatory that the incidence of spinal injuries in children increased. These injuries include abdominal ecchymosis mirroring the lap belt, intra-abdominal soft tissue injury, and lumbar flexion-distraction fractures. Anatomic considerations that lead to an increased risk of the seat belt syndrome in children are as follows: the increased head-to-body ratio results in a higher center of gravity and increased movement occurring about the axis of rotation, the relatively underdeveloped rib cage leaves the majority of abdominal organs unprotected, and the small pelvis does not support the belt. The lap belt is displaced superiorly to the level of the lumbar spine, as the passenger submarines beneath it during a head-on collision (Fig. 52-2). L3 is the most commonly fractured vertebra with this mechanism.

More than 75% of flexion-distraction fractures occur as a result of the use of a lap belt, and the incidence has mirrored legislation in safety requirements for children in motor vehicles. It has also contributed to the current guidelines developed by the American Academy of Pediatrics for weight-based safety seating for children. Now children who are at risk for "seat belt syndrome" are required to be placed in booster seats with a broader lap restraint and a chest strap.

Spondylolysis deserves some discussion in this chapter on lumbar spine injuries in children because it occurs as a result of an overuse syndrome. It is thought to be caused by repetitive trauma to the pars interarticularis, resulting in a stress fracture of the posterior elements and pain. Most patients, when questioned directly, remember a particular game or episode when the pain first started. For children who play soccer, this often was after a high-velocity kick.[3] Spondylolysis can result from flexion overload, unbalanced shear forces, forced rotation, and repeated flexion-extension.[12]

TYPES OF INJURIES

Lumbar spine injuries range from paravertebral soft tissue sprains to compression fractures, transverse and spinous process fractures, and three-column flexion-distraction injuries. Most have different mechanisms of injury and are managed according to severity. We discuss the nonsurgical and surgical management of each in the following sections.

PARAVERTEBRAL SOFT TISSUE SPRAINS

Some patients who present after traumatic incident have paravertebral soft tissue injuries without injury to the spinal column. In one study, the incidence was reported to be approximately 68% of all injuries in the spinal region.[2] These injuries fall into the realm of strains and sprains, and the children complain of either neck or back pain in the setting of negative imaging studies. As with fractures, because of the larger head-to-body ratio in a child, the paracervical region is most commonly affected. Lumbar sprains account for 5%
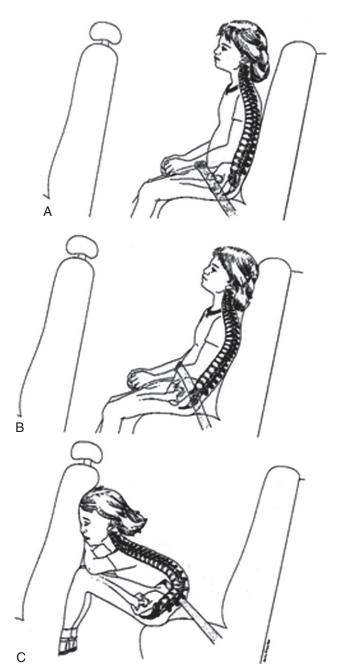
Fig. 52-2 Mechanism of injury with lap belts. *A,* Correctly positioned belt and child. *B,* Incorrectly positioned belt with slouching child. *C,* Simulated head-on collision and flexion-distraction of spine. *(Reproduced with permission from Johnson DL, Falci S: The diagnosis and treatment of pediatric lumbar spine injuries caused by rear seat lap belts. Neurosurgery 26:434–441, 1990.)*

of traumatic soft tissue injury and occur in children who are older than 6 years.[2] Soft tissue injuries, such as a lumbar sprain, are made as a diagnosis of exclusion after traumatic episode. In these situations, the child, even in the setting of negative-imaging studies, will still complain of back pain.

These soft tissue injuries also occur as a result of participation in sports. In a prospective study of 550 elite and nonelite athletes in Australia, 32% of those who sustained injuries had injury to the lower back. Most of the injuries occurred secondary to hyperextension injuries.[13] Of these, most had no structural abnormalities detected and were thought to be soft tissue injuries alone. Sports such as gymnastics, football, and wrestling also have been associated with lumbar sprains.

These injuries are managed conservatively with a brief period of rest and anti-inflammatory medication and then rehabilitation if necessary. Return to activities is encouraged in the absence of any other significant injury.

STABLE FRACTURES

Occasionally, fractures of the lumbar spine do not affect the overall stability of the spine. Single column injuries, as defined by Denis,[14] can be managed nonsurgically although they do contribute to morbidity because of associated pain. These stable fracture injuries include transverse process fractures, spinous process fractures, and fractures of the pars interarticularis and lamina.

Isolated transverse process, spinous process, and lamina fractures often are the result of fairly significant blunt trauma. The presence of these fractures should prompt the evaluating physician to look for other associated injuries. These fractures alone do not require surgical intervention but might indicate that a more significant injury is present at another level in the spine or within another organ system. MRI studies of the whole spine have revealed that the incidence of a secondary level, noncontiguous fracture is approximately 34%.[15] At this time, it is not practical or even cost-effective to perform whole-spine MRI if a transverse process fracture is identified; however, the clinician must perform a detailed physical examination with a visual inspection and palpation of the posterior elements and paraspinal area, and if there is any concern, further imaging is warranted. Transverse process fractures also have significant association with abdominal injury; if identified, CT should be performed to rule out visceral injury.

COMPRESSION FRACTURES

Compression fractures of the vertebral body are classified by Denis[14] as *major* spinal injuries. They are, however, considered stable if less than 50% loss of height occurs between the anterior and posterior body. Any more compression of the anterior body leads to an increased likelihood that the posterior ligamentous structures have failed. Relatively good outcomes can be achieved when these fractures are managed nonsurgically. Some use bracing of the spine with a lumbar orthosis. Lower levels are difficult to completely immobilize without the use of a leg extension; however, a leg extension secondarily can be used as a gentle reminder to the child that he or she has an injury that must be protected for a period of time. Long-term follow-up of patients who sustained compression fractures during early adolescence shows that approximately 80% will be pain free in the region of injury with virtually full restitution of height and rarely any deformity. The remaining 20% experience only occasional back pain and are not significantly disabled.[16]

BURST FRACTURES

Burst fractures occur less commonly in children than in adults because of the greater mobility and elasticity of the pediatric spine. Burst fractures are two-column injuries that involve retropulsion of the vertebral body into the canal. Canal compromise can lead to significant neurologic injury. This fracture is unstable and leads to progressive kyphosis, and morbidity is left untreated. Children who sustain burst fractures can develop mild progressive angular deformity at the site of the fracture. Operative stabilization prevents further kyphotic deformity and decreases the length of hospitalization without contributing to further cord compromise. Nonoperative treatment of a burst fracture is a viable option in neurologically intact children, but progressive angular deformity can occur during the first year after the fracture.[17] This involves progressive ambulation in a full thoracolumbar orthosis or cast, with added leg extension if the fracture is below L3. The cast is maintained for approximately 12 to 24 weeks, depending on symptoms and radiographic evidence of healing. Surgical correction involves anterior decompression and fusion.

FLEXION-DISTRACTION INJURIES

Chance[18] first described this fracture pattern in 1948 in a report of three cases. His description was primarily of a bony injury involving fracture through the ossified elements of all three columns. Many, including Gumley,[19] Gertzbein,[20] and Court-Brown,[21] have since described and categorized this injury, some including soft tissue ligamentous disruption. It was Denis[14] in 1983 who described it as a three-column injury caused by flexion and distraction of the spine. The mechanism of injury is a fulcrum bend of the spine around a fixed point. This leads to flexion of the anterior body and distraction of the posterior elements. The anterior longitudinal ligament is thought to be the anterior tether and fulcrum bend.

The fracture pattern helps to guide management. A true Chance fracture is more likely to achieve bony union if satisfactory sagittal alignment can be maintained with nonsurgical methods, such as casting. The method of closed reduction and casting is performed with the child under sedation. Hyperextension is used to reduce the fracture, and a cast is applied. If the fracture is below L3, a unilateral leg extension is recommended to help immobilize the lumbopelvic region. As mentioned earlier, abdominal injury is significantly associated with lumbar chance fractures. Injury requiring surgical intervention might contraindicate early casting. In these cases, if the child remains neurologically stable, he or she should be placed on bed rest until abdominal injuries have been managed. If injuries are so severe that multiple laparotomies are required, surgical stabilization with instrumentation could be considered.

If the injury primarily involves the ligamentous structures posteriorly, a risk of nonoperative treatment is the development of late junctional kyphosis caused by an incompetent posterior tether. In such cases, a surgical approach is preferred, with stabilization of the posterior tension band. Only the vertebrae above and below the posterior ligamentous injury need be involved in stabilization. A variety of instrumentation can be used, including pedicle screws and laminar hooks. Preoperative CT is essential to evaluate the pedicle and posterior elements for fractures. Because this fracture also commonly occurs in the school age population, evaluation of pedicle size is advised to help select an appropriate screw diameter.

RING APOPHYSEAL FRACTURES AND DISK HERNIATIONS

Ring apophyseal fractures occur between the ring apophysis and the cartilaginous rim of the vertebral endplate. This ring develops in the child between the seventh and ninth years of life and represents ossification of the peripheral portion of the cartilaginous endplate. It fuses to the vertebral body by 18 years of age. This fracture involves displacement of the apophysis posterior into the canal and often is associated with a concomitant disk protrusion (Fig. 52-3). Patients often present with leg and back pain and have signs and symptoms that overlap those of an acutely herniated disk, such as radiculopathy. The posterior ring of L4 is the most common location, and approximately 30% of cases are associated with lumbar Scheuermann disease. The four subtypes of vertebral endplate fractures are as follows: type I, pure avulsions of the posterior cartilaginous endplate;
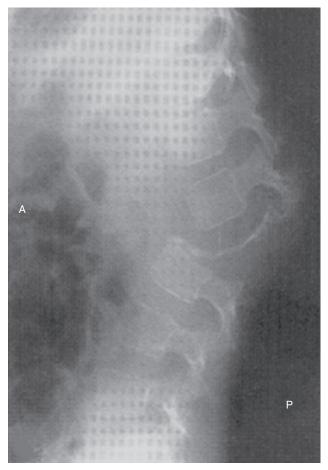

Fig. 52-3 Female patient with ring apophyseal fracture.

type 2, lesions that have large central fractures and include part of the bony rim; type 3, fractures localized posterior to an irregular cartilage endplate; and type 4, fractures that involve the entire posterior portion of the vertebral body. Common activities that cause this fracture include shoveling, hyper-extension activities, and weight lifting. Treatment involves laminotomy and excision of the loose fragments and decompression.

True disk herniations have been documented to occur in the pediatric population but rarely occur in children younger than 10 years. Initial management is conservative; however, if symptoms fail to improve, posterior diskectomy is indicated. Percutaneous endoscopic diskectomy has proved to be a reasonable option to open techniques in the management of herniated disks.[22]

SPONDYLOLYSIS

Symptomatic spondylolysis in the absence of significant listhesis can be managed conservatively with a 3-month period of rest and then slow return to activity. If the patient fails conservative management for more than 6 months, our treatment protocol is to perform a diagnostic pars injection under radiologic guidance. If this area is the primary pain generator and if symptoms are completely relieved by the injection, we proceed with a posterior approach and fixation of the pars fracture. The fracture can be fixed with lag screws across the defect itself, pedicle screw and hook constructs, Scott wiring techniques, or modified Scott wiring. Many cadaveric studies have tested the strength of each construct; however, to date, no long-term prospective studies have evaluated clinical outcome based on construct type.

The management of a symptomatic spondylolysis with spondylolisthesis depends primarily on the grade of slip, angle of slip, and presence of neurologic symptoms. Meyerding grade I and II slips can be managed as previously discussed. The patient should adhere to a period of rest and then a slow return to sporting activity once symptoms improve. If more than 50% translation is present at presentation, the likelihood of progressive slip is high and such cases rarely respond to conservative management alone. In addition, patients with a kyphotic slip angle and/or symptomatic nerve root compression require surgical intervention. Posterior fusion supplemented by anterior interbody fusion has shown the best long-term results for high-grade slips. The decision of whether to reduce high-grade slips remains controversial. Reduction helps to restore sagittal alignment and assist with nerve root decompression, but nerve root injury from traction is a risk.

CONCLUSIONS

Acute lumbar spine fractures are rare in the pediatric population. MVAs cause the majority of these injuries, and concomitant involvement of other organ systems, primarily gastrointestinal, is likely. The head-to-body ratio of a child makes the risk of cervical injuries more likely. Sports participation also is associated with significant lumbar injuries in children. Most of these injuries, whether the result of traumatic injury or overuse syndrome, can be managed

nonoperatively for good results. When surgery is indicated, knowledge of the anatomy of the developing spine is essential for evaluation of preoperative imaging studies and selection of appropriate implants.

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C H A P T E R

53

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Sacral Fractures in Skeletally Immature Patients

INTRODUCTION

Although sacral and posterior pelvic ring fractures in the adult population have a bimodal distribution affecting patients subject to high-energy injury trauma and an elderly population with metabolically impaired bone, skeletally immature patients are fortunately rarely affected by such injuries.[1,2] Aside from patients affected by collagen and other primary skeletal dysplasia variants, the lumbosacral junction is relatively well protected and rarely injured in a pediatric or adolescent population. Specific concerns to a skeletally immature population sustaining traumatic disruption of this area mainly revolve around injury survival and long-term effects of growth impairment. Insights into this injury entity in an adult population are difficult to come by because of sporadic incidence.[3] This is a far more accentuated problem in a pediatric population with much lower occurrence compared with their adult counterparts. Any discussion of treatment options in a pediatric population requires an understanding of the relevant anatomy as well familiarity with a variety of treatment options available for a variety of injuries. In addition, familiarity with the development of the posterior pelvic ring and spinal column is helpful to aid in treatment decisions.

INCIDENCE

Pediatric sacral fractures are unusual injuries. The mainstay of literature consists of case reports or concentrates on posterior pelvic injuries.[1,4–13] In a retrospective review by Hart et al.[14] in 2004, of 4876 cases of pediatric trauma at one children's hospital over a 7-year period, there were eight sacral fractures. At our trauma center over the past 15 years, we have treated 436 pelvic ring injuries in those 18 years old and less, and 131 in those younger than 13 years.

DEVELOPMENT

Knowledge of the ossification centers and dates of occurrence, as well as consolidation of the pediatric lumbosacral region and pelvic structures, is a prerequisite for management of injuries for diagnosis and management of injuries in this age group. During fetal growth the sacrum ossifies at 8 weeks, and at 2 to 8 years the posterior elements and vertebral bodies fuse.[15] At 20 years of age, the secondary ossification centers have closed. At 7 years, the arches fuse with the vertebral bodies. As the child grows, sagittal balance is maintained through increasing the pelvic tilt and lumbar lordosis.[16,17] The ossification centers of the sacrum are shown in Figure 53-1, *A* and *B*.[18] Similar to adults, the sacrum dictates the later alignment of the lumbar spine through its sacral inclination angle.[12] Certain fracture patterns may predelict a patient for later back pain secondary to excessive pelvic incidence.[16]

EVALUATION

Because it takes exceptional forces such as motor vehicle crashes, falls from a height, or deceleration trauma to disrupt the lumbosacral structures in a healthy pediatric population, basic trauma care principles should be applied to any and all such patients suspected to have sustained pelvic trauma.[1,3,4,11,19,20]

High-energy trauma often results in multiple injuries involving the head, chest, spine, abdomen, and extremities.[21,22] The primary assessment goal is to attend to the resuscitation needs of the patient and monitor the patient responses thoughtfully. As in an adult patient, systematic circumferential head-to-toe clinical evaluation is performed with inspection and palpation. Important findings relevant for lumbopelvic trauma include ecchymoses, swelling, tenderness, and presence of a ballotable posterior fluid accumulation (Morel-Lavallee lesion).[23] Because of its crucial relevance for an occult open-pelvic fracture, the examining physician should also check for blood at the meatus of the urethra and from the vagina or rectum. Displaced sacral fractures of the pelvic ring may coincide with considerable blood loss, especially through the sacral venous plexus and arteries such as the superior gluteal artery. Emergent countermeasures to this
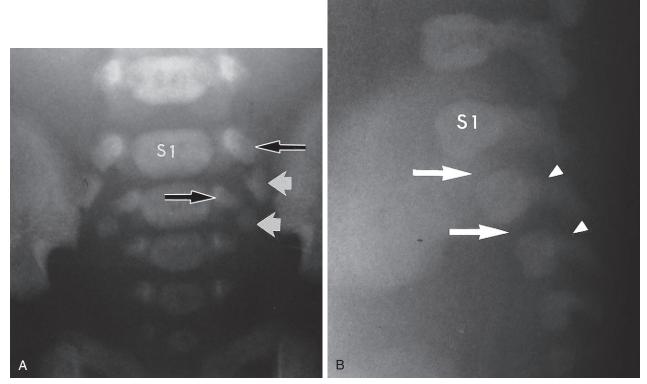
Fig. 53-1 Sacrum in a newborn. *A,* Frontal radiograph shows two of the three ossification centers *(black arrows)* that form the sacral ala. The central ossification centers contribute to the vertebral bodies of the sacrum (S1 to S5). The costal ossification centers *(white arrows)* form a portion of the lateral mass. *B,* Lateral radiograph shows the intervertebral disk spaces *(arrows)* of the sacrum. The neural ossification centers are seen posteriorly *(arrowheads).* (From Diel, J, Ortiz, O, Losada, RA, Price, DB, Hayt, MW, and Katz, DS: The sacrum: pathologic spectrum, multimodality imaging, and subspecialty approach. Radiographics 21(1):83-104, 2001.

scenario may consist of application of compressive pelvic sheeting or angiographic embolization.[24] Neurologic assessment of patients with spinal injury requires assessment of lower sacral root function through directed assessment of motor, reflex, and sensory function, just like in an adult. This delicate examination has to be performed with due consideration for age and maturity of the patient at hand and usually benefits from clear explanation to the patient regardless of age, and to parents, and should be conducted in the presence of experienced pediatric or trauma nursing staff. Systematic documentation, just like in any adult patient, should be performed, and repeat evaluations should be carried out depending on the patient's status and injury. In no way should a neurologic status be "inferred" in a pediatric patient with spinal injury by conjecture with subsequently management by assumptions.[2]

IMAGING

The basic principles of spinal and pelvic ring imaging well established for adults should be applied to injured pediatric patients without deviation. Aside from spinal imaging studies and anteroposterior pelvis radiographs, the conventional imaging of the pelvic ring is accomplished with an anteroposterior (AP) pelvis, inlet, outlet, and sacral lateral views. For smaller pediatric patients often a "pan-torso" AP radiograph encompassing chest, abdomen, and pelvis is used as a screening study. It should be considered, however, that an AP pelvis radiograph has a low sensitivity in diagnosing pelvic abnormalities.[5,19] The sensitivity is even lower in children, with a reported 33.3% sensitivity in detecting sacral injuries and 75.5% sensitivity for diagnosis of pubic bone fractures.[19] The advent of helical computed tomography in an emergency room setting has produced a thorough paradigm shift in the imaging approach toward severely injured patients because the entire patient trunk can be rapidly imaged. In a nonintubated pediatric patient of questionable compliance with lengthy imaging protocols, greater imaging speed can allow for valuable imaging information to be gained without having to resort to sedation, or even intubation, for imaging purposes. The onus has thus been turned on the managing clinician to ask for appropriately reformatted images in case of suspected lumbopelvic injuries. Although dedicated fine-cut computed tomography (CT) may still hold a resolution advantage over spirally acquired images, this imaging modality

should be reserved for truly complex injuries in pediatric patients because of radiation concerns. Key diagnostic images for a pediatric sagittal and coronal reformats usually are helpful as well as sufficient in evaluating sacral fractures.

Pediatric CT radiation dose had and is being looked at closely by the radiology community in the last few years[25] (http://www.cancer.gov/cancerinfo/causes/radiation-risks-pediatric-CT). Typically, children would receive a greater radiation dose because their bodies are smaller than adults if scanned at the same settings as adults. This would represent a factor of 1.0 to 2.5 times greater radiation dose depending on the age of the child. However, most modern CT scanners have software that can adjust the imaging settings by age and weight of the patient and thus could optimize dose settings to a patient's best interest. For instance the effective dose typical for an adult pelvis ranges from 7 to 10 mSv. A child's radiation dose should be lower because the technical factors can be adjusted. The American College of Radiology recommends the pediatric (5-year-old) abdomen radiation dose CTDI weighted to 25 mGy.

Key diagnostic images for a pediatric sagittal and coronal reformats are usually are helpful in evaluating sacral fractures. Helpful hints as to the severity of the initial trauma can be gleaned from the size of peripelvic hematoma seen on the initial pelvic CT scan.[26]

OTHER DIAGNOSTIC MODALITIES

MRI

The use of magnetic resonance imaging (MRI) in a young pediatric patient is commonly a complex undertaking in regard to the technicalities involved. In clinical reality, most pediatric patients require intubation and a general anesthetic to minimize motion artifact and make an MRI feasible. As much as there may be theoretical advantages in obtaining pelvic MRI in the workup of patients with patent growth zones, in reality this imaging modality plays a very limited role in the workup of sacral fractures in general and in pediatric patients specifically. Typical indications for MRI remain limited to workup for stress fracture, suspected malignancy, or infection. From a trauma standpoint, visualization of neural elements in the sacrum is rarely useful for clinical decision making. Discongruent clinical levels of neurologic and skeletal levels of injury are one of the rare instances in which a sacral MRI is desirable. Routine MRI for sacral fractures—as in adults—is, however, not necessary.

ELECTRODIAGNOSTIC STUDIES

As in adults, EMG and somatosensory evoked potentials (SEPs) may be helpful in assessing neural element injuries, especially in patients with diminished clinical compliance.

Anal sphincter may show lower sacral root injuries, however, with a latency of several weeks following initial insult. In practical reality, this test is virtually impossible to obtain in an awake pediatric patient.[2] For suspected lower sacral root injuries pudendal Somatosensory Evoked Potentials (SSEP) can, however, provide real-time feedback as to presence of injury. Similar to adult patients with cauda equina deficiency, long-term bladder function can be assessed with postvoid residuals or cystomyography. Fortunately, the occurrence of neurologic injury in the sacrum in a pediatric population is exceedingly rare.[2]

SACRAL FRACTURE CLASSIFICATION

In general, sacral fractures may occur in conjunction with, or independent of, posterior pelvic ring trauma. It is important for the treating physicians to assess the patient's injury structurally as to the impact on pelvic and lumbosacral stability.[27] Historically, classification systems suggested for pediatric posterior pelvic ring fractures have been based on a limited number of anecdotally observed factors, such as disruption, as presented by Torode and Zieg[28] in their system for pediatric posterior pelvic ring injuries. Similar to the somewhat unsystematic multifold systems historically presented for adults, we suggest consolidating classification attempts based on the location of the most medial fracture extension relative to the neural canal because of its strong correlation with neurologic injury in a logically progressive severity scale.[29] Because there is no difference between the pediatric and the adult biostructure in terms of neuroskeletal relations, we see no cogent reason to implement separate classifications for adult and pediatric patients. The key variable presented consists of the degree of skeletal ossification and the extent of injury to the cartilaginous and growth plate "anlage" (rudimentary tissues). For purposes of stratification of general sacral ring injury, the Denis sacral fracture classification remains applicable as in adults.[30] Zone I fractures are lateral to the neuroforamina, with a 5% or less rate of L5 injuries reported in an adult population. In contrast type II injuries have their most medial fracture line extend through the neuroforamina in 15% to 30% of patients, mainly with L5 and S1 root injuries.[30] Zone III injuries involve the spinal canal and have a more than 50% incidence of cauda equina deficits. Subdifferentiation using the Roy-Camille system with Strange-Vognsen supplementation can help quantify the severity of the sacral trauma.[31,32]

Classification of neurologic injuries affecting the sacral plexus has been proposed by Gibbons et al.[33] Undoubtedly this system has shortcomings for adults, such as ignoring impairment of sexual function. In light of sacral root recovery potential with unilateral or partial lower sacral root preservation, clear documentation of any deficits for comparison to later outcomes remains an important decision-making help for definitive treatment.[34]

EMERGENT TREATMENT OF SACRAL FRACTURES

As previously stated, sacral fractures usually are the result of a high-energy mechanism and therefore necessitate the appropriate workup and resuscitation. Airway, breathing, circulation, and exposure are first assessed and adequately addressed, usually under the direction of the emergency room physician or traumatologists. Important input for early resuscitation efforts can be provided by a specialist familiar with pelvic trauma, by recognizing injury patterns typically associated with potential for large-volume losses and suggesting countermeasures.[6,10,18,20] Spinal precautions should be maintained during the trauma evaluation and workup, until satisfactory evidence that there are no noncontiguous is obtained spinal injuries. Any patient who presents with a spinal injury must have imaging of the entire spine because the incidence of noncontiguous injuries can be quite high.[35]

For patients with hemodynamic instability and concurrent pelvic ring injuries, the cause of bleeding from the sacral venous plexus or actual arterial damage from the superior gluteal artery should be considered.[4,23] In these instances, a simple sheet extending from the iliac crest extend to just below the great trochanter and secured anteriorly in a snug fashion can help in "closing" and stabilizing the pelvic ring injury and allow for hemostasis. A percutaneously applied external fixator may serve the same purpose.[24] Consideration should strongly be given to angiography of the pelvis to locate the bleeding source and embolize it, especially when hemodynamic stability cannot be achieved, and there are no other apparent sources. Commercially available so-called pelvic clamps are usually unsuitable for small children because of fixateur dimensions that are not appropriate or safe for application in pediatric patients with their generally smaller pelvic bony elements. Moreover, there are other less invasive means of greater or equal effectiveness for accomplishing pelvic volume reduction and temporary fixation.
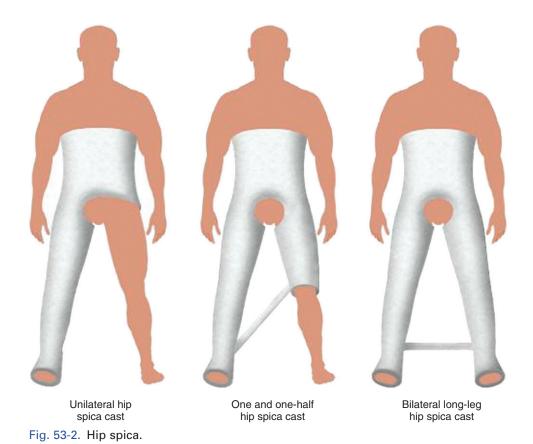
TREATMENT OF PEDIATRIC SACRAL FRACTURES

The collective published literature to date overwhelmingly recommends nonoperative management of pediatric sacral and posterior pelvic fractures.[1,5,6,9,11,20,36–39] Most authors agree that nonoperative therapy should focus on keeping pelvic asymmetry to less than 1 cm of displacement because of its higher propensity for leg-length discrepancy, scoliosis, back pain, and limp as undesirable outcomes.[16,27,39,40] Although it is incompletely understood, a traumatically induced increase of a sacral inclination angle may cause considerable long-term disability through its effect on spinal alignment.[16] Therefore, it seems reasonable to pursue an adequate sacral and posterior pelvic ring reduction and maintain it, whether the child is treated operatively or nonoperatively.

Protected weight bearing, if the child can follow the restrictions, may be used for minimally displaced fractures. In those instances in which greater stability is preferred, a hip spica brace may be used, or a hip spica cast may be used in children who are too young to comply with restrictions and in those whom greater stability is required (Fig. 53-2). Unilateral pelvic ring disruptions may be treated with closed reduction and the use of recumbent skeletal traction, followed by a hip spica extended below the knees. Cognitively nonimpaired children will often self-protect an injury and will start crawling when they have achieved painless healing of their injury. A pantaloon cast, which extends from the mid chest to just proximal to the knees, provides stability for injuries that involve both sides of the pelvis, as well as in those instances in which more rigid immobilization is needed, without clear indication for surgery. Mobilization of patients may have to be minimized during the period of extensive casting and bracing and the may have to be limited to wheelchairs with a reclining back. There are few if any specific recommendations for the duration of nonoperative or recumbent treatment for pediatric patients with pelvic ring or sacral fractures. Depending on the age of the patient, a period of recumbence ranging between 3 and 8 weeks should suffice to allow for initial consolidation of a fracture. Follow-up bracing for completion of a 3- to 4-month period following injury should suffice to allow for remodeling with minimal risk of secondary loss of alignment. Anticoagulation should be individualized depending on age and size of the patient affected.[41]

SURGICAL TREATMENT

Fortunately, surgical treatment of skeletally immature patients with sacral fractures is rarely indicated. Accepted surgical indications revolve around open sacral fractures and injuries with neurologic compromise, as well as in patients with unacceptable lumbosacral malalignment.[8,13,35,42–44] It is commonly assumed that pediatric fractures may remodel, but this may not be sufficient to correct a significant deformity, especially when an injury affects predominantly ligamentous structures or in adolescents with minimal residual growth. As previously mentioned, pelvic deformity is not readily tolerated and may have unknown long-term consequences. Without question late corrective measures are highly invasive procedures with considerable potential for complications, even in experienced hands. Hence an argument for early surgical intervention within 3 weeks can be made for selected patients in whom early closed reduction is unsatisfactory.[45]

Conceptually, surgical treatment in a pediatric patient population ideally uses the least invasive procedures suitable to achieve and maintain injury reduction combined with some temporal activity restriction. Anterior pelvic external fixation may be used with the pins placed in the iliac crests just next to the anterior-superior iliac spines. Percutaneous screws may be placed over a guidewire under fluoroscopic guidance from the posterior ileum into the sacral ala.[46,47]

Unilateral hip
spica cast

One and one-half
hip spica cast

Bilateral long-leg
hip spica cast

Fig. 53-2. Hip spica.

Screw diameters adapted for patient size may be chosen, such as 4.5-mm screws for preadolescent individuals.[46,48] The most extensive treatment option for posterior pelvic ring disruption and high-grade sacral ring disruption consist of open neural element decompression, open reduction, and iliolumbar segmental fixation. Historically, these surgical techniques have used nonrigid techniques modified from the Galveston technique initially used for pediatric spinal deformity surgery.[49] Direct internal fixation options of the posterior pelvis are highly complex procedures necessitating some degree of improvisational skills while increasing the risk of soft-tissue breakdown and limited biomechanical stability.[50] Therefore, we have found these procedures to be generally undesirable in lumbosacral trauma patients because of inherent limitations in reduction and structural stability. Techniques for comprehensive segmental stabilization of even highly complex and displaced injuries in an adult population are described in the pertinent chapters for adult fracture sacral fractures and are conceptually very similar in the pediatric population.[41,51–53] Successful restoration of all aspects of lumbopelvic alignment, with resumption of full weightbearing as tolerated, without rigid bracing and satisfactory neurologic recovery rates in presence of sacral plexus injuries, with relatively low complication rates, have been reported for adults.[23,54] In contrast to adults, hardware removal should be strongly contemplated in pediatric patients 6 to 12 months

after surgery.[51] To date there no larger series of comprehensive lumbosacral fixation used for pediatric sacral or lumbosacral disruptions, aside from case reports.[13,43]

TREATMENT RESULTS IN LITERATURE

Trauma accounts for more than one half of pediatric deaths in children age 1 to 14 years old, primarily from motor vehicle injuries.[20] Pediatric pelvis trauma is relatively rare, comprising 2.4% to 7.5% of pediatric fractures.[39] There have been two studies reporting the relative distribution of types of pelvic injury in mature and immature patients[21,39] (Table 53-1). The incidence of sacral fractures is extremely low in the pediatric population, and reports in the literature reflect the dearth of knowledge on this topic. The literature contains primarily case reports: a S1-S2 traumatic "sacrolisthesis" in a 13-year-old boy with neurologic deficits who was treated initially in traction to reduce the fracture and later with posterior stabilization, which resulted in some neurologic recovery[12]; an S1 fracture-dislocation in a 12-year-old boy who underwent a closed reduction and recovered motor, bowel, and bladder function 13 months after the injury[8]; and a zone III sacral fracture and a fracture dislocation in two children, an 8 and a 13 year old, who did not have neurologic deficits and were treated conservatively without

TABLE 53-1   **Distribution of Pediatric Pelvic Fractures**

AUTHOR/SOURCE	N	LOCATION/TYPE OF FRACTURE PATTERN	MATURE* (%)	IMMATURE† (%)
Silber, et al.[21]	133	Ramus	31	53
		Iliac wing	29	6
		Acetabulum	6	44
		Pubic/SI diastasis	5	19
Beckenstudie II (personal correspondence with author)[39]	42	Type A‡	61.8	52.6
		Type B§	27	31.3
		Type C‖	21	18.4
		Acetabulum	12	0

*Triradiate cartilage closed.
†Triradiate cartilage open.
‡AO/Tile: iliac wing and border fracture without ring compromise.
§AO/Tile: rotational instability with SI joint disruption.
‖AO/Tile: pelvic ring disruption with rotational and vertical instability.

fracture reduction.[7] In a more recent 2004 retrospective study by Hart et al.[14] found 8 cases of sacral fractures in 4876 pediatric traumas over a 7-year period (0.16% of all pediatric trauma and 4.76% of children with pelvicfractures). All pediatric sacral fractures resulted from motor vehicle accidents or falls from height and half were associated with a pelvic ring injury. Six had a Denis zone I injury, one had a zone II injury, and one had a zone III injury and was the only one that sustained a significant neurologic injury. This child underwent a decompressive laminectomy and had some improvement following surgery. Two case presentations from our institution demonstrated the potential for management of complex unstable lumbosacral injuries with concurrent neurologic and soft tissue injuries. The pediatric ability to remodel bone and recovery initially hopeless appearing neurologic injuries in high-grade sacral fracture cases underscores the value of having comprehensive surgical reconstruction techniques available.[13,34,43]

CONCLUSION

Despite an exceedingly rare occurrence, sacral fractures in a pediatric population should not be ignored. Especially with greater availability of high-speed recreational sports activities to ever-younger individuals, clinical suspicion of sacral fractures has to be at a level commensurate to the injury history and mechanism. Increasingly larger children, increasingly more high-energy injuries from a variety of sources, and general diagnostic unfamiliarity pose a potential for missed sacral injuries. Despite younger children's innate greater ability to remodel bone deformity and recover from neurologic deficits with nonoperative therapy compared with surgical intervention, awareness for the potential of injury has to remain high. Missed injuries bear a disproportionate burden of substantial pathology. Pertaining to the eventual choice of care, however, each case, should be assessed based on predicted stability, residual deformity, and neurologic compromise with an eye on the overall injury burden of the patient following a systematic injury evaluation.

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Complications Associated with Surgical Management of Pediatric Spine Trauma

INTRODUCTION

Every invasive procedure performed across surgical specialties has the potential for various complications. In orthopedic surgery, a specialty with which fixed internal hardware is the norm, complications can be related to the surgical procedure itself or to the implanted hardware. Spine surgery is no exception. Spine surgery uses various plate and screw constructs, in addition to metal wires and external fixators to achieve fixation in both elective and emergent procedures.

Complications of spine surgery in the adult population have been well described in the available literature. They include pin-site problems with the use of halo fixation, superficial or deep surgical site infection, and problems related to failure of the implanted hardware. In the pediatric age group, however, the literature is sparse. Studies describing the complications of pediatric spine surgery in general are limited to a few publications at best; regarding pediatric spine trauma, the number is even smaller. In an attempt to discuss the complications of pediatric spine trauma surgery in the face of little published data, it is necessary to extrapolate from the data published regarding adult spine trauma surgery and the data published relating to elective spine surgery, namely adolescent idiopathic scoliosis, spondylolysis, and spondylolisthesis. This chapter focuses on the complications encountered in pediatric spine trauma surgery and their management.

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COMPLICATIONS RELATED TO PATIENT POSITIONING

In addition to the surgery itself, patient positioning for spine surgery is one of the most formidable tasks a surgeon can face. Considering that many of the operations involve the prone or knee-on-chest position, great care must be taken to avoid pressure points that can cause pressure necrosis, peripheral nerve palsy, compartment syndrome, or venous thrombosis.

The most common complications occurring from improper positioning involves neurologic injury. In the adult population, the most common neurologic injury involves the brachial plexus, which has accounted for up to 38% of neurologic injuries in one study.[1] Other peripheral nerves that are vulnerable during positioning include the ulnar nerve at the elbow, often subjected to tension injuries, and the radial nerve at the wrist. Proper padding of these areas and placing the arms in extension can help minimize complications. Additionally, placing pillows under the ankles of the patient in the prone position can help to avoid problems associated with traction on the sciatic nerve.

COMPLICATIONS RELATED TO HALO SKELETAL IMMOBILIZATION

The halo skeletal fixator provides the most rigid fixation of all cervical orthotic devices.[2] The overall complication rate has been reported to be as high as 68%,[3] but the vast majority of the complications tend to be relatively minor, involving pin loosening and pin-site infection.

Garfin et al.[4] and Lind et al.[5] reported pin loosening occurring in as many as 36% to 60% of adult patients. In the absence of infection, they recommend retightening the pins to 8 in-lb in adults and to 2 to 5 in-lb in infants and children as long as resistance is met during the first few revolutions. If no resistance is met, however, a new pin should be placed in an alternative location before removal of the loose pin.[4]

Pin-site infections have been reported to occur in up to 20% to 22% of patients.[4,5] Treatment of this complication involves administration of oral antibiotics and local pin care

in most cases but might require parenteral antibiotics and even incision and drainage if cellulitis or abscess is present.[4]

Other complications associated with halo immobilization include pressure sores, which occur in 4% to 11% of patients[4,5]; pin site bleeding, which is a rare complication that occurs in only approximately 1% of patients[4,6]; dysphagia in 2%, which often is the result of overextension and resolves with repositioning[4]; loss of reduction, which results in redislocation in up to 10% of patients[5,7–10]; and dural puncture, which is a rare complication resulting most often from falls or other trauma and requiring administration of antibiotics, removal of the old pin after placement of a new pin, and sometimes surgery.[4,6,11]

BLEEDING COMPLICATIONS ASSOCIATED WITH SPINE SURGERY

Blood loss is a complication known to be associated with all spine surgery, generally increasing in magnitude in proportion to the size of the spinal segment involved. Several surgical techniques can be optimized to decrease total blood loss during spine surgery; one of the most important is the speed of the surgery. One study suggests that the use of intraoperative tranexamic acid in pediatric patients undergoing scoliosis surgery can reduce blood loss by up to 41%.[12] The authors recommend a bolus dose of 100 mg/kg before incision and then a maintenance dose of 10 mg/kg/hour during surgery. Methods to decrease the amount of blood transfused for spine surgery include preoperative autologous donation, intraoperative blood salvage techniques, and intraoperative hemodilution,[13] as well as hypotensive anesthesia.

INFECTION

One of the most frequent causes of morbidity associated with spine surgery is infection. Reported to occur in approximately 1% to 2% of all cases of spine surgery, infection is the most frequent condition requiring return to the operating room for additional procedures. Several studies have focused on identifying infection rates in the adult trauma population, but the literature is much sparser regarding pediatric patients. One study evaluating arthrodesis of the cervical spine for fracture and dislocation in children and adolescents identified a superficial infection rate of 2%.[14] Fortunately, all the infections were superficial, did not penetrate the deep fascia, and required only superficial irrigation and débridement. Similar infection rates were obtained by other investigators who evaluated all pedicle screw complications experienced by patients younger than 18 years undergoing thoracolumbar and lumbar fusions. The authors noted one deep wound infection in their series that required hardware removal.[15] The overall infection rate associated with spine surgery remains fairly low, with most infections being superficial, requiring superficial irrigation and débridement.

BONE GRAFT HARVEST SITE COMPLICATIONS

Complications related to bone graft harvest at the iliac crest are numerous and can be severe. They include infection, neurovascular injury, and continued postoperative pain and numbness. Fortunately, the overall incidence of harvest site complications is low, reported to be 2%.[16]

Neurovascular injury is an infrequently reported complication associated with bone graft harvest site. Skaggs et al.[16] reported a 0.5% incidence of arterial injury at the sciatic notch in a retrospective review of 214 children who underwent spinal fusion with posterior iliac crest bone graft. This same group also reported complications observed much more often, including continued postoperative pain and numbness. The incidence of continued pain at the harvest site was reported to be 24%, with 15% reporting pain that interfered with daily activities. Nine percent of the children required daily nonsteroidal anti-inflammatory drug therapy. Additionally, 20% complained of numbness around the scar.

Superficial infection after iliac crest bone graft harvest has been reported to occur with an incidence of 0.5% to 2%.[14,16] Skaggs et al.[16] noted that the majority of the infections were superficial, requiring a short course of intravenously administered antibiotics and single irrigation and débridement. Fortunately, the infection rates are low; however, they cannot be overlooked when discussing the morbidity of this procedure.

The complications and complication rates associated with surgical management of pediatric spine trauma are similar to those associated with surgical management of adult spine trauma. In the adult population, the most frequent complications involve infection, pain, and damage to the superior cluneal nerves and the superior gluteal artery.[17]

COMPLICATIONS RELATED TO HARDWARE AND INSTRUMENTATION

A wide range of complications can occur related to the placement of spinal hardware and instrumentation. For the most part, the complications can be generalized as being related to malposition of instrumentation or technical error. The complications are broad and range from screw malposition and neurovascular injury at the time of surgery to loss of fixation and progressive deformity after surgery.

Several studies presented in the trauma and the scoliosis literature reported the incidence of neurovascular injury in pediatric spine surgery. Neurovascular injury during surgery for idiopathic scoliosis has been reported to occur in approximately 1% of patients of all ages. A closer look at data from 1995 reveals that of 1643 patients undergoing surgery for idiopathic scoliosis, one developed a complete spinal cord

defect and ten developed partial spinal cord and nerve root injuries.[18] The etiology of these complications has implicated a wide range of possible causes, including excessive traction on neural elements, direct trauma, contusion, and vascular insufficiency. Regarding vascular injury or insufficiency, one study recommends ligating vessels at the mid-vertebral body–level, rather than at the neural foramen, or temporary clamping if possible.[19] Neurologic injury secondary to pedicle screw malposition in cases of pediatric spine trauma is a rare event. A review of 223 cases involving 759 thoracolumbar and lumbar pedicle screws in patients younger than 18 years revealed that short-term complications occurred in five patients, ultimately leading to 17 screw removals.[15] Of the five patients, only two had lumbar radicular complaints: one with bilateral L3 to L5 pedicle screws had right quadriceps weakness postoperatively and recovered normal strength 8 weeks after screw removal; another with T4 to L3 pedicle screws complained of left thigh pain but no weakness postoperatively. This resolved when the screws were removed 6 months later.[15]

In addition to neurovascular injury, pedicle screw placement can cause other problems in the pediatric population. A study examining pedicle screw placement in 1- and 2-year-old children with various pathologic abnormalities reported complications that included pedicle fracture, screw breakage, and failure of screw connection.[20] The authors also commented on screw malposition involving three of 91 pedicle screws in this population; fortunately, no symptoms of neurologic abnormality occurred.[20]

Postoperative complications in the pediatric population relating to hardware placement and fusion masses have also been evaluated. The complications include spontaneous extension of the fusion mass in up to 38% of patients, incorrect level of fusion with rates reported to be as high as 2%, incomplete fusion masses, and progressive kyphotic deformity after surgery.[14,21]

CONCLUSION

The complications associated with spine surgery in the pediatric population are many and can occur at all times during the perioperative and postoperative course. By knowing the complications that can occur with any particular treatment, a surgeon can develop methods for avoiding them. Strict attention to detail in patient positioning preoperatively can decrease the incidence of nerve and skin complications. Proper application and care of external fixation devices, such as the halo immobilizer, can minimize the complications of pin loosening and infection. Expeditious surgery combined with hypotensive anesthesia can minimize surgical blood loss. Meticulous attention to detail with the placement of instrumentation can limit complications associated with its insertion. Rapid recognition of superficial postoperative infection can head off the development of more serious infective complications. Only through

further research and study of the treatment of pediatric spine trauma can we further reduce the overall incidence of perisurgical complications.

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Pediatric Spinal Column and Spinal Cord Injury and the Use of Luque Segmental Instrumentation in the Injured Pediatric Spine

INTRODUCTION

The vast majority of spinal column and spinal cord injuries occurs in adults, with only approximately 10% of these injuries involving children.[1,2] The differences in the prevalence of pediatric and adult spinal injuries are multifactorial, including less exposure to high-energy trauma during childhood and increased flexibility of the pediatric spine. A more flexible spine can dissipate energy better and, thus, potentially reduce the risk of traumatic injury in children. At the same time, however, the immaturity of the spinal column also produces a unique spectrum of spinal injuries, such as a greater propensity for ligamentous, upper cervical spine, and spinal cord injury without radiographic abnormality (SCIWORA). In this chapter, we review traumatic spinal injuries in pediatric patients and the use of Luque (rod and sublaminar wire) instrumentation to treat these injuries.

OVERVIEW OF TRAUMATIC SPINAL INJURIES BY AGE

The underdevelopment of ligamentous structures and facets within the cervical spine results in hypermobility in normal pediatric patients as compared with adults. The cervical facet joint, an important stabilizer of the adult spine, has a horizontal orientation in the developing spine, as opposed to a more vertical orientation in adults. The horizontal orientation of the facets, underdevelopment of the uncovertebral joints, and greater ligamentous flexibility allow for greater sagittal plane translation in the pediatric cervical spine. Normal age-related hypermobility can be appreciated on cervical spine radiographs with the phenomenon of pseudosubluxation, in which cervical vertebral bodies can translate up to 4 mm in normal children, and the observation of greater atlantodental intervals in children than in adults.[3,4]

In addition to hypermobility, the size of a child's head relative to his or her muscular development produces different injury patterns during childhood development. Spinal injuries during childhood can be broken into three unique age groups: (1) those occurring in infants, (2) those occurring during early childhood (children from the age of head control to 8 years), and (3) those occurring in late childhood (children of approximately 8 to 15 years).[5] Injuries to the infant spine are rare and are most often the result of birth trauma.[6-9] Most injuries occur in the high cervical spine, above C4, and are associated with variable degrees of neurologic injury.[10] Injuries to the thoracolumbar spine occur less often.[11]

Early childhood injuries are most often secondary to motor vehicle accidents, falls, or abuse and, like those in infancy, tend to affect the upper cervical spine as the more unstable biomechanical features of this region act in concert with a relatively large head, which creates a high fulcrum of rotation.[12-15] Thoracolumbar injuries are often seen in cases of abuse, but their overall incidence remains low in comparison with to injuries of the cervical spine.[16,17] Another unique injury pattern seen in this group is the Salter-Harris fracture, or fracture through the growth plate, which is considered highly unstable and is often associated with neurologic injury.[18-20] Fractures may also occur through the odontoid subchondral synchondrosis and neurocentral synchondrosis.[21,22] These fractures can easily be overlooked on plain radiography and likely contribute to the types of injuries previously described as SCIWORA. With the advent of magnetic resonance imaging (MRI), however, they are often detected.[23]

The spinal column of the child older than 8 years is similar to that of the adult in morphology and biomechanical stability.

Thus, the injuries in these age groups are similar to those seen in adults, with cervical injuries affecting the middle-lower cervical spine and a greater frequency of bony injury. Salter-Harris type fractures are no longer seen and the incidence of SCIWORA is greatly reduced. Injuries in this group are more common than in the younger groups as a result of greater activity levels.[1,2] The most frequent mechanism of injury between the ages of 10 and 15 years is sports related, with motor vehicle accidents placing a close second.[1,2]

CLINICAL MANAGEMENT

CLINICAL PRESENTATION AND EXAMINATION

The clinical presentation of a child with a spinal injury is highly variable, and history should provide the first clue to the presence of a spinal injury. Because a considerable amount of force is needed to injure the spinal column, injuries to the spine are associated with other injuries, including cardiac contusions, pneumothoraces, abdominal injuries, and head injuries, approximately 50% of the time.[2,16] The index of suspicion should be also be raised in patients who are uncooperative or in those who display indirect signs of spinal trauma, including seat belt marks, facial or head lacerations, bruises, crepitus, or a palpable gap between the spinous processes.[20,24,25] Certain mechanisms of injury, such as abuse, high-level falls, and motor vehicle accidents, are associated with greater risk for spinal injury and should prompt a thorough evaluation. Finally, autonomic dysfunction, specifically hypotension without tachycardia, should alert the examiner to the possibility of spinal shock.[25]

Precautions to maintain spinal alignment should be initiated on all patients during initial evaluation by emergency medical technicians or other medical personnel. Specific attention must be give to younger children (age <8 years) who have relatively large head (occiput)-to-body ratio because placement on a standard backboard may force the neck into kyphosis and contribute to fracture malalignment.[26] Apediatric-specific backboard with an occipital cut-out or the placing of an extra pad beneath the body can be used to maintain anatomical alignment.

After the patient is stabilized, a complete neurologic examination should be performed, along with a thorough visual and tactile inspection of the spine. Rectal tone and the presence of the bulbocavernosus reflex should be documented if a spinal cord injury is suspected. Radiographic examination should be prompted by any abnormal exam finding, significant pain or guarding, or any of the previously listed findings.

DIAGNOSIS OF PEDIATRIC SPINE INJURY

Adequate imaging is critical in the workup of spinal fractures. Numerous pitfalls exist in the imaging evaluation of children because of their unique anatomy and biomechanics. Infants are difficult to assess with plain radiography because a large portion of their spines are cartilaginous and most injuries sustained are ligamentous in nature.[27] Plain radiographs, however, do constitute the beginning of most evaluations and are valuable in assessing alignment and fractures.[28] Assessment of odontoid fracture, soft-tissue swelling, and subluxation can be difficult in children. A review of the use of transoral x-rays in the assessment of odontoid injuries indicated that they have little utility in children younger than 9 years.[28] The unossified synchondrosis in young patients can additionally be confused with fracture. Children may have increased width of the prevertebral soft-tissue window when crying, which can make it difficult to distinguish from pathologic swelling related to an injury. The assessment of instability may also be difficult with dynamic radiographic imaging because children may exhibit pseudosubluxation and may have an atlantodental interval of up to 4 mm on flexion views as normal variants.[3,4] Finally, the assessment of the integrity of the craniocervical junction may be complicated by variable ossification of the odontoid that makes its relationship with the skull base difficult to ascertain. Additionally, measurement such as the Powers ratio may miss dislocations at this joint that are posteriorly displaced or have reduced with external stabilization.[29]

Computed tomography (CT) scanning has greater utility in the assessment of fractures and enables a more accurate examination of fracture morphology and canal compromise, although care must be taken not to confuse ossification centers in younger children with fractures. CT evaluation, however, is inadequate in the detection of ligamentous injuries, which are the most common type of injury in children younger than 10 years.[30,31] MRI is the best modality to assess for injury of the soft tissues and nervous tissue in children. MRI evaluation should be used whenever a child presents with a neurologic deficit because it may reveal soft-tissue, ligamentous, or disk injury not detected with other modalities.[32] Its use may be invaluable in the evaluation of SCIWORA and in the evaluation of obscure injuries such as atlanto-occipital dislocation or odontoid fractures that are not well visualized with plain x-rays or CT.[21] MRI evaluation may also provide prognostic information in the case of spinal cord injury.[33] Finally, MRI may be used to evaluate and clear the cervical spine of a child who is comatose or unable to cooperate with an examination.[27]

NONOPERATIVE MANAGEMENT

Although the pediatric spine is predisposed to injury for a number of reasons, surgical indications and techniques specifically designed for children are lacking because the injuries themselves occur infrequently. Additionally, most pediatric spinal injuries can be managed nonoperatively with external immobilization because the pediatric spine has a greater propensity for healing than the adult spine.[13] Even ligamentous injuries in children

have the potential to heal with nonoperative management.[34] Despite more frequent success with the nonoperative option, however, stable external fixation of the spine can be difficult in children because of their small body habitus, flexible spinal columns, and poor cooperation in young children.[13] Additionally, nonoperative treatment may delay mobilization and predispose the patient to infections or vascular, pulmonary, and psychiatric morbidity.[35,36] Finally, halo immobilization, a mainstay in the treatment of cervical injuries, is associated with a significant complication rate in children and may inhibit participation in rehabilitation.[37,38]

INDICATIONS AND CONTRAINDICATIONS FOR SURGERY

Advances in surgical techniques and instrumentation and a greater awareness of the potential complications of immobilization devices have led to greater interest in pediatric spinal surgery in cases of trauma.[39] Early internal fixation enables the patient to mobilize more rapidly and participate in rehabilitation, decreasing the overall length of hospitalization and reducing complications.[35,36] Current indications for surgical spinal stabilization include irreducible or progressive deformities, decompression of neural elements, and grossly unstable injuries.[13]

When surgery is indicated, the use of internal fixators is often required for stabilization. The concept of internal spinal fixation was originated by Lange[40] in 1910, but it did not gain wide acceptance until Harrington began to develop his internal fixation system in the 1950s.[41] Favorable initial results in the treatment of scoliosis led to utilization of internal fixation in the treatment of degenerative disease and instability caused by tumor, infection, and trauma.[42] The Harrington system did have significant problems, however, including poor rotational stability, the need for external orthoses, difficulty in low-lumbar and sacral fixation, and difficulty in maintaining normal curvatures of the spine with the resultant creation of flat back syndrome and associated problems.[42,43]

Luque instrumentation, developed by Eduardo Luque in Mexico in the 1970s, was also designed to be used for the correction and stabilization of scoliotic deformities. The Luque technique involves the segmental attachment of bilateral contoured rods to the spine with sublaminar wires.[44] In scoliosis procedures, the sublaminar wires are gradually affixed to the contoured rods as correction maneuvers are performed. In addition to its use in thoracolumbar deformity surgery, the Luque system has been used in a similar fashion for the treatment of instability from the occiput to the sacrum.[45,46] Revisions in design have led to the creation of an L-shaped rod with a bend on one side that is inserted through the interspinous space, further securing the construct, and the development of the Galveston technique, by which the rods are inserted into the iliac wings for pelvic
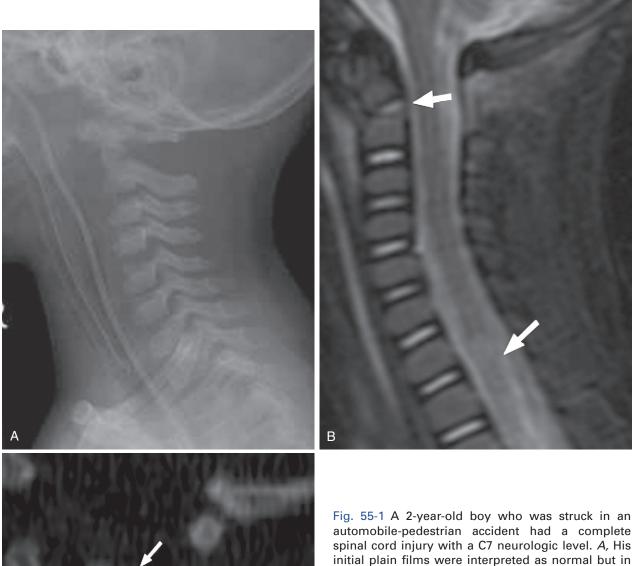
fixation.[45] The firm fixation at multiple levels with sublaminar wiring provides a stronger construct with fewer hardware failures than Harrington rods and allows for early mobilization while minimizing the need for postoperative orthoses.[45,47–49] The passage of sublaminar wires is reported to be safe in children after the age of 6 when their spinal canals reach adult proportions.[50]

Although the Luque system was a significant step forward, it had several drawbacks. Because the rods are smooth and connected to the spine with wires, there exists a potential for the rods to telescope through the wires and allow the spine to compress or shorten. In addition, biomechanical testing has shown that the construct is not capable of applying compressive or distractive forces.[51] The nonrigid fixation offered by this system also allows for load sharing, which, although useful in promoting bone healing in the stable spine,[52] is a detriment when attempting to prevent vertebral collapse. Additionally, it has been recommended that long fusion constructs (three segments above and below the unstable segment) be used in cases of instability[53]; however, although this might be acceptable in the thoracic spine, it is associated with flat back syndrome, decreased mobility, and long-term painful sequelae when used in the lumbar spine.[54] The technique of "instrumenting long, fusing short," in which the rods are removed after fusion at the level of interest is achieved, has been proposed to address this problem but mandates a second operation for instrumentation removal and a compliant patient.[53] Finally, the immobilization of joints may additionally result in their arthritic degeneration or even autofusion.[55,56]

The use of sublaminar wires has additionally been associated with a significant rate of neurologic injury, reported to be as high as 13%.[57] Fortunately, most of these injuries are minor, consisting of hyperalgesia, and resolve on their own within a month's time.[57] The rate of major neurologic injury is much lower—just 0.4% in one series of 512 cases.[58] Cerebellar infarction has also been reported with the use of wires and Luque instrumentation in occipital-cervical stabilization.[45] Finally, the removal of sublaminar wires in revision surgery adds an additional element of danger as the wire tips cannot be controlled.[59]

Since the introduction of Luque instrumentation, numerous advances have been made in spinal stabilization. Segmental screw fixation, with either pedicle or lateral mass screws, has become the most commonly used method of spinal instrumentation and provides numerous advantages over previous techniques. The increased biomechanical strength of screw fixation has enabled the fusion of fewer segments with greater stability, the improved maintenance of normal spinal curves, and the preservation of more functional spinal units.[54,60–62] Although superior in most applications, the use of pedicles screws may be difficult or impossible in children with small pedicles,[63] and these cases may necessitate the use of sublaminar

wiring, especially in the thoracic spine. Furthermore, sublaminar wiring may be better suited than screws for applications of occipital-cervical fixation when the calvarium is thin.[64] When using Luque instrumentation in traumatic applications, however, the surgeon should be cognizant that this instrumentation is unable to provide distractive or compressive forces and is thus unable to prevent vertebral collapse. Thus, although it may be suitable for the treatment of three-column translational injuries and some flexion/compression fractures, it should not be used without anterior reconstruction for burst or severe compression fractures (Fig. 55-1).

Fig. 55-1 A 2-year-old boy who was struck in an automobile-pedestrian accident had a complete spinal cord injury with a C7 neurologic level. *A,* His initial plain films were interpreted as normal but in retrospect showed some angulation of the odontoid process. *B,* MRI studies showed spinal cord edema and hemorrhage at the cervicothoracic junction *(white arrow)* and increased signal in the odontoid synchondrosis *(red arrow). C, On* CT images, there was widening *(white arrow)* of the odontoid synchondrosis as depicted in the sagittal reconstructed images shown.

OPERATIVE PROCEDURE AND TECHNIQUE

OPERATING ROOM SETUP

Operating room preparation for the use of Luque instrumentation is similar to that for most spine cases. Before surgery, preoperative antibiotic therapy is administered to the patient. After intubation, neural monitoring consisting of both somatosensory and motor evoked potentials is set up and baselines are recorded. Monitoring should be strongly considered in patients without complete injury because the spinal cord is at risk not only from the injury itself but also from the passage of sublaminar wires. Care is taken to maintain spinal alignment while rotating the patient into the prone position. A Jackson table is used because it allows for optimum positioning to maintain or achieve reduction while also allowing for easy use of fluoroscopy. Once the patient is positioned, nerve potentials are reconfirmed and the fluoroscopy is used to guide in marking the segments of interest. The use of Luque instrumentation requires long-segment constructs, with fixation of three levels above and below the unstable level usually being recommended.

EXPOSURE

Exposure of the spine is undertaken with a standard midline incision. The soft tissues are dissected from the bony elements in a subperiosteal manner. The entire length of the spine to be instrumented should be exposed laterally to the transverse processes. After bony exposure is obtained, the spinous processes in the thoracic spine are removed with a double-action rongeur to allow for access to the ligamentum flavum in the midline. The spinous processes in the lumbar spine can be left in place if adequate exposure can be obtained with resection of the interspinous ligament alone. The facets of the levels to be fused and the transverse processes are decorticated at this point to facilitate fusion. The laminae should be left intact because they provide the strength for securing the wires and their removal offers little, if any, advantage in wire passing.[65] The ligamentum flavum can then be removed with a Kerrison rongeur or sharply with a knife and a Penfield dissector used to protect the thecal sac.

PLACEMENT OF INSTRUMENTATION

Instrumentation begins with the placement of sublaminar wires. Two double wires should be used at the inferior and superior extents of the construct and at segments in which additional strength may be needed. Proper wire contour should be obtained before passage of the wires. We advocate using a semicircular shape with a radius of the curvature at least as large as the width of the lamina because this configuration has been shown to provide less canal penetration than others.[65,66]

The wires are passed in a caudal-to-cranial direction in the midline. The shaped double end of the wire is carefully inserted under the lamina and advanced forward until the tip reaches the superior aspect of the lamina. Light dorsal traction should be maintained to keep the wire taut against the inner surface of the lamina and to minimize intrusion. A rolling, circular motion in the shape of the curve of the wire is then used to pass the curved portion of the wire through the interspinous space of the above segment. A Kocher clamp or a hook can be used to pull this portion of the wire through until approximately equal lengths of wire are exposed on either side of the lamina. Any resistance to passage of the wire should be dealt with by withdrawing the wire and re-attempting passage, after confirming midline position. The tip of the double-ended wire is cut and the wires are crimped on either side of the lamina for stability. The strands of the now-separate wires are secured to opposite sides of the transverse processes. The tails are brought out of the wound laterally in such a way that they will not be prominent and dislodge during the remainder of the procedure.

Next the rods are bent to fit snugly against the laminae of the aligned spine. A standard three-point rod bender is adequate for most traumatic applications, although a bending unit can be helpful for longer fusions. The rods are designed so that the horizontal part of one L-rod crosses the interspinous space at the superior aspect of the wound and the other at the inferior aspect, creating a box configuration. The superior strand of the wire is brought over the rod while the inferior strand is left underneath. The two strands are twisted until tight, indicated by a change in color from shiny to dull gray. After the entire rod is secured, all of the wires should be retightened. Cross links may be added for additional rotational and translational stability. Copious amounts of bone graft are then placed over the decorticated transverse processes to provide substrate for a fusion mass, and the wound is closed in the normal fashion (Fig. 55-2).

GALVESTON TECHNIQUE

The Galveston technique was developed by Allen and Ferguson[48] as a method to provide stable pelvic fixation in scoliotic patients. Although this technique can be used for trauma, it has been largely replaced by more rigid screw fixation to the sacrum and pelvis. In the Galveston technique, exposure of the iliac wings is accomplished through a midline incision to include the posterior superior iliac spine, the midportion of the iliac wing, and the superior portion of the sciatic notch. The insertion point is just posterior to the sacroiliac joint at the level of the posterior superior iliac spine. A notch in the bone can be created at this point to facilitate instrumentation. A Steinmann pin of equal diameter to the rod is used to create a tunnel that will accept the rod. The pin should be directed approximately 1.5 cm above the sciatic notch with care taken to keep the pin between the cortices of

the ilium. A length of rod of at least 6 cm should be inserted into the pelvis. The pin is then removed, and a malleable template is used to aid in shaping the Luque rod, which is then placed into the holes and attached to the remainder of the spine as previously described.

OCCIPITAL-CERVICAL FUSION

Although atlanto-occipital dislocation is often fatal, advances in prehospital care have brought more of these patients to the attention of providers in good condition.[67] Advances in instrumentation have additionally enabled the rigid fixation of the craniocervical junction in children with transarticular screws and an occipital plate.[68] Although such fixation is biomechanically superior to fixation with sublaminar wires, enables the sparing of subaxial motion segments, and often obviates the need for postoperative halo stabilization,[68–71] the operation is technically demanding and not suitable for all anatomical variations. Furthermore, concern has been expressed about the use of

screw fixation in a thin occiput, and therefore wiring might be the optimal choice in these cases.[64]

Occipitocervical fusion in these patients begins with careful placement of the patient in the prone position. A Mayfield 3-point fixation device is used to provide rigid stabilization if the child is old enough. Proper alignment is confirmed with fluoroscopy after positioning. A midline incision is created from just below the external occipital protuberance to just below the lowest spinal level to be included. It has been recommended that two motion segments (lamina) below the level of pathology be wired.[46] The muscles are then dissected off the occiput and the exposed laminae in a subperiosteal fashion. Rongeurs and dissectors are used to remove soft tissue and define the extradural space between the laminae of interest. Three burr holes are then created in the occiput, one in the midline and two above and lateral to the superior border of the instrumentation. Sublaminar wires are then passed as detailed above while two double wires are passed from the lateral to medial burr holes in the occiput. The Luque
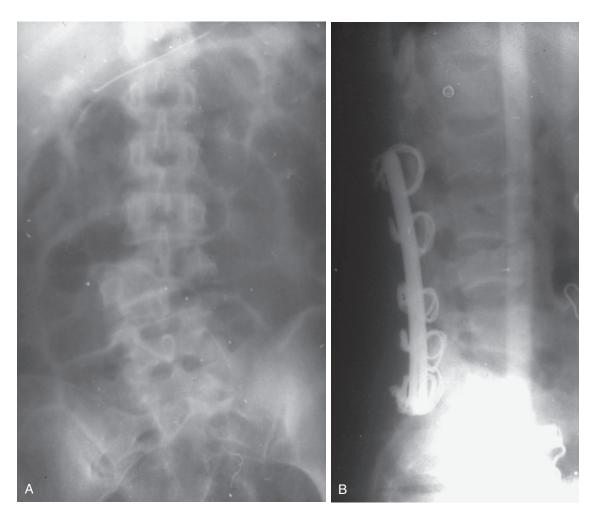

Fig. 55-2 *A,* Anteroposterior radiograph of an L3-L4 fracture dislocation that was treated with Luque technique. *B,* Postoperative lateral anteroposterior radiograph showing sublaminar wiring with Luque rods from L2 to sacrum.
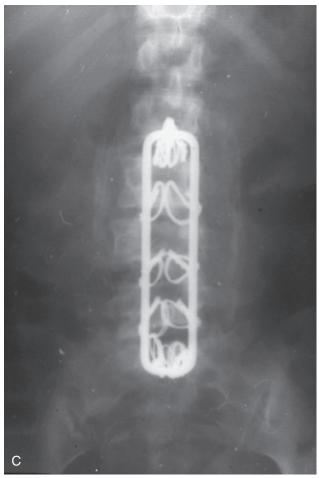
Fig. 55-2 *C,* Anteroposterior view.

rods are then contoured to fix the occiput in anatomic alignment and are fastened to the bony elements with the wires as described previously (Fig. 55-3). Cross links can be added to increase torsional stability in longer constructs. The wound is closed in the normal fashion.

COMPLICATION AVOIDANCE

Complications associated with Luque instrumentation include neurologic injury,[51] infection,[57] pseudarthrosis,[57,71] and hardware complications such as breakage of both wires and rods and erosion through the skin.[72,73] The most alarming of these is neurologic injury, which most often consists of transient paresthesias or hyperalgesia but can rarely result in significant deficit.[57,58] We recommend the use of neural monitoring in all patients without complete injury. We also recommend careful bending and passing of wires as detailed above to produce minimal canal intrusion.[66]

Infection has been reported to occur in between 4%[71] and 15%[57] of patients treated with Luque instrumentation and may be higher than in patients treated with other instrumentation constructs because of the duration of these complex procedures.[54,71] Avoidance of infection involves the use of standard operating room sterile technique and preoperative administration of antibiotic agents.[74] The wound should be thoroughly irrigated before closure, and consideration should be given to the use of a dilute Betadine solution washout to prevent infection.[75,76] Infections may need to be treated with drainage and débridement and prolonged antibiotic therapy, but the hardware and graft can usually be left in place and most patients go on to achieve solid fusion.

Wire breakage is also a frequent complication,[59] albeit one usually without major consequence.[72] The greatest stress is experienced at the ends of the constructs, and double wires are used in these locations for this reason. The ends of the double wires should be wired to the rods to prevent bothersome protrusion. Rod breakage occurs rarely and is associated with pseudarthrosis, the use of smaller ($^3/_{16}$ in.) rods, and failure to correct deformities.[72,73] One-quarter–inch rods are thus recommended, and every effort should be made to restore anatomic alignment.

Medical complications, including pulmonary complication, urinary tract infections, blood clots, and gastrointestinal

Fig. 55-3 *A,* Photograph of Luque rods that could be used in posterior cervical stabilization. *B,* Double-stranded wire is created with a bend in the wire and placed sublaminar for fixation to the rods.

problems, can be minimized with early mobilization allowable in the setting of multitrauma.

SURGICAL OUTCOMES

A pediatric series evaluating the use of Luque occipital-cervical stabilization is lacking from the literature. Good fusion results have been achieved in series of adults, but neurologic complication rates of up to 20% has been reported. These complications have included cerebellar infarctions and quadriplegia, which may be related to the passage of sublaminar wires.[46,77] Thus, the complication rate of the procedure and the decreased biomechanical stability it offers in comparison to that of screw fixation make Luque craniocervical stabilization a second-choice procedure.

CONCLUSION

Pediatric spinal column and cord injury is rare and is composed of a unique spectrum of injuries secondary to the changing biomechanics of the developing spine. Most injuries can be treated nonoperatively because the immature spine has a greater propensity than the adult spine to heal. Operative stabilization is warranted, however, when significant instability places neural elements at risk or when nonoperative treatments are likely to fail.

Luque instrumentation, first described for use in correcting scoliotic deformities, has been effective in stabilizing the injured spine from the occiput to the sacrum. The technique, however, has the drawbacks of requiring relatively large constructs, a risk of neurologic injury with the passing of sublaminar wires, and the inability to apply compressive or distractive forces. These shortcomings have led to its use having been largely supplanted by more modern means of screw fixation, which have greater biomechanical stability and allow for the fusion of fewer segments. Nevertheless, screw fixation can be difficult to perform in the pediatric thoracic spine with small, immature pedicles and in stabilization of the craniovertebral junction where the occiput may be too thin to achieve good purchase, and the placement of atlantoaxial screws can be technically demanding. In these instances, the use of Luque instrumentation remains a viable alternative.

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DAVID M. WALLACH

Pedicle Screw Fixation in the Pediatric Spine: Anatomic Considerations and Techniques for Placement of Screws in the Thoracic, Lumbar, and Sacral Spine

INTRODUCTION

Children are not adults in miniature. Anatomic, physiologic, and behavioral differences distinguish the child from the adult. Body proportions (head size to body ratio), open synchondrosis, facet orientation, ligamentous laxity, and poor muscle control account for the different injury mechanisms and patterns observed in children.[1–3] Perhaps as important as the physical differences that affect patient care are the emotional and cognitive differences attributable to immaturity. Often, children require external support to "slow" them down and protect their fixation.

Even with the previously mentioned challenges, children by in large do well.[4] The comorbidities that so often plague the adult spine patient, such as diabetes, steroid use, intravenous substance abuse, and smoking, occur less often with children. The healing potential of children is phenomenal. High fusion rates are seen even without the use of autogenous bone.[5,6] This
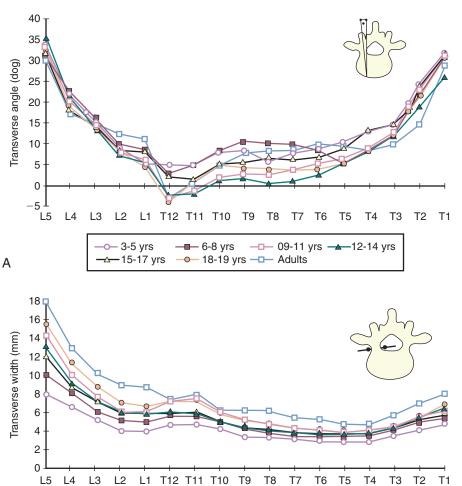
is not to say that children would not benefit from the advances made in adult spinal surgery. Pedicle screw use offers many advantages in the treatment of the pediatric spine. Screws provide three-column support,[7] enhanced rotational control (an issue in spinal deformity surgery), lower profile than hook constructs (in the lumbar spine), no canal intrusion when well placed (important in the congenitally stenotic spine),[8] and the theoretical benefit of epiphysiodesis without an anterior fusion to prevent crank-shaft phenomenon.[9]

Our understanding of pedicle anatomy, improved screw designs, and methods of screw insertion have changed greatly since 1948 when King[10] placed the first screw into a pedicle. The screw technique used by King consisted of a trans-facet screw that engaged the pedicle for improved purchase. It was not until 1969 that screws were placed down the axis of the pedicle.[11] Pioneers of pedicle screw systems, including Steffee et al.,[12] Roy-Camille et al.,[13] and Cotrel et al.,[14] developed methods of connecting the screws to plates and/or rods.

ANATOMY

Pedicle morphology (Fig. 56-1) was described for adults initially by Saillant[15] and later in English by Vaccaro et al.,[16] Zindrick et al.,[17] and Scoles et al.[18] The transverse outer cross-sectional diameter of the pedicles is largest at L5, typically measuring 15 mm or more, with a gradual decrease to 8 mm at L1. The transverse diameter is smaller than the sagittal diameter (an oval). The diameter of thoracic pedicles decreases as one ascends from T12 (approximately 7 mm) to T4-T5 (approximately 4 mm) and then increases again in diameter as one ascends to T1 (approximately 8 mm). The medial inclination is approximately 35 degrees at L5, with an approximate 5-degree decrease as one ascends to L1. The 12th thoracic pedicle can angle laterally 5 degrees. Medial inclination stays at approximately 5 to 10 degrees of angulation from T10 up to T4. Medial inclination then increases up to 26 degrees at T1.

In a follow-up article, Zindrick et al.[19] evaluated pedicle morphology in children. He found similar angle measurements for directing screws. Predictably, pedicle size varies by age. For the lower lumbar spine, pedicular width was an average of 5 mm or more in children in the youngest age group

Fig. 56-1 Transverse pedicle angle *(A)* and transverse pedicle width *(B)*. (From Zindrick MR, Knight GW, Sartori MJ, et al: Pedicle morphology of the immature thoracolumbar spine. (25:2726–2735, 2000.)

measured (3 to 5 years old). Senaran et al.[20] showed that the pedicles in children who were 5 to 8 years old were large enough at L4-L5 to accept standard implants but usually required "custom" implants at higher levels to accommodate an inner dimension of 2.3 mm.[20] Adequate pedicle size (for noncustom implants) was not reached until 6 to 8 years of age in the upper lumbar and lower thoracic spine. It should be noted, however, that Ruf and Harms,[21] in a clinical series, showed that even the pedicles of toddlers could accept standard adult cervical spine screws (Fig. 56-2). Mid-thoracic pedicles do not reach a diameter of 4 mm until adolescence. Pedicle length (from entrance of pedicle to anterior cortex of vertebral body) at L5 measured 30 and 50 mm, respectively, for 3- to 5-year-old children and adults. A slow and gradual decline in pedicle length to 22 and 37 mm, respectively, at T2 occurs for 3- to 5-year-old children and adults.

The studies described previous pedicular anatomy in the nondeformed spine. When a rotational deformity is present, the pedicle is further reduced in size.[22,23] Furthermore, defor-

mity causes the neural tissue to migrate closer to the concave pedicles (Fig. 56-3). In such cases, the use of an extrapedicular technique (in the thoracic spine) has advantages.[24] The rib-pedicle unit, as described by Husted et al.,[25] increases the effective transverse diameter of the pedicle (Fig. 56-4). When the pedicle is narrow and lacks an endosteal center, the vertebral body is entered at the base of the pedicle where it joins the vertebral body. No attempt is made to cannulate the pedicle.[26] The extrapedicular technique has comparable biomechanical strength with the transpedicular techniques.[27]

Multiple structures are at risk during pedicle screw placement. Within the canal is the dura, spinal cord, and exiting nerve roots.[28] Suk et al.[28] reported 67 malpositioned screws among 4604 screws. Complications directly related to screw placement included one pneumothorax, one paraparesis (resolved), and three durotomies. Soyuncu et al.[29] reported that the nerve roots of adult spines could come within 1.29 mm of the pedicle. Ugur et al.[30] found that the nerve root can be as close as 0.8 mm from the inferior aspect of the thoracic
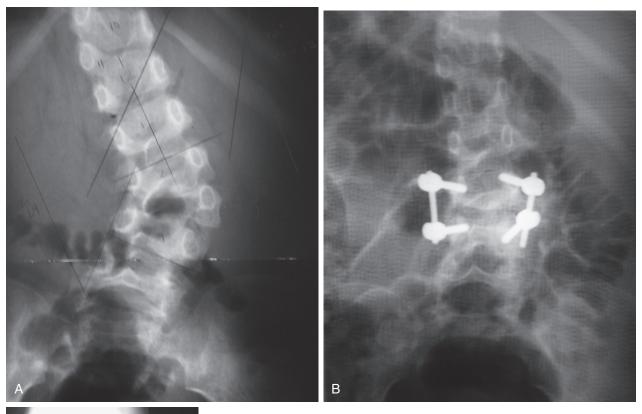
Fig. 56-2 *A*, Preoperative PA image of a 5-year-old boy with congenital scoliosis. *B* and *C,* Postoperative PA and lateral images following transpedicular excision and pedicle screw instrumentation using 3.5-mm cervical implants.
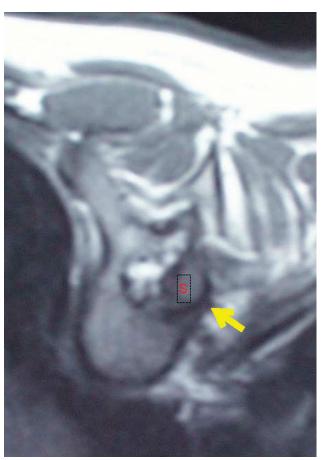
Fig. 56-3 An axial MRI image of a pediatric spine taken through the apex of the scoliosis. Note the migration of the spinal cord *(S)* toward the smaller pedicle *(arrow)* located on the concave side of the curve.
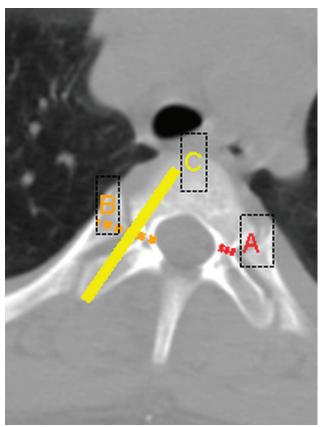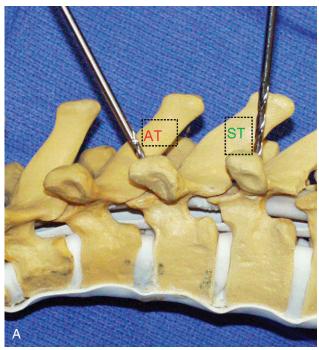

Fig. 56-4 An axial image of the upper thoracic spine. *A,* The pedicle width. *B,* The combined width of the pedicle and rib head. *C,* The trajectory of an extrapedicular screw.

pedicle. Anterior to the vertebral body are the esophagus, aorta, vena cava, azygos vein, sympathetic chain, and lung.[16,31,32] Structures at risk when using sacral screws include the middle sacral vessels, the common iliacs, the L5 nerve root, and the intestine.[33] Fortunately, low complication rates have been reported in association with screw placement in children.[34]

BIOMECHANICS

Steffee et al.[12] coined the term *force nucleus* to highlight the importance of the pedicles. The pedicle is located at the convergence of the lamina, facets, transverse process, and muscle insertions (at the adjacent mammary and accessory process). It is through the pedicle that all posterior forces are directed to the front of the spine. Early studies showed the biomechanical superiority of the pedicle screw over hooks and wire constructs.[35,36] Improved corrections have been reported in clinical series comparing screws with hooks for the treatment of spinal deformity.[37]

For the thoracic spine, two options for screw placement in the pedicle are available (Fig. 56-5): the anatomic technique

(AT) and the straight-ahead technique (ST). The superiority of the ST over the AT was shown by Lehman et al.[38] Using a cadaveric model, they showed that screws placed with the ST had 39% greater torque on insertion and were 27% stronger on pull-out than those placed with the AT. The AT can be used as a "bail-out" when the medial wall is violated during straight-ahead screw placement. In a subsequent article, Lehman and Kuklo[39] showed that conversion to the anatomic trajectory (must use a polyaxial screw) will achieve 62% of the maximal insertional torque (torque achieved when placing the ST method). Another bail-out is to use the extrapedicular technique. With this method, one can achieve 64% of the biomechanical strength of an all-inside technique.[40] This more lateral position also requires polyaxial screws and makes it more challenging to interpret intraoperative radiographs regarding medial pedicle wall violations.

Screw length directly affects purchase strength. Krag et al.[41] showed, in a cadaveric model, that an increase of length from 50% to 80% (of the vertebral body) resulted in a 32.5% increase in pull-out strength. Penetration through the anterior cortex results in an additional 20% to 31% increase in pull-out
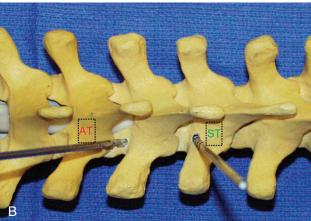
Fig. 56-5 Orientation and placement of thoracic screws using the anatomic *(AT)* and straight ahead *(ST)* techniques as seen from the lateral *(A)* and posterior *(B)* views.

strength depending on screw diameter.[42] Selecting the optimal screw length for a particular vertebra is therefore a balance between the improved grip strength afforded by longer screws and the increased risk of injury to structures anterior to the vertebra. With the exception of sacral screws, all screws should be no longer than 80% of the vertebral diameter (as discussed later).[43]

Anterior cortical purchase is desirable when placing sacral screws to offset the biomechanical disadvantage of the short pedicle length (37 mm at S1 vs. 50 mm at L5) and the thin cortical shell of the "sacral pedicle."[42,44] Unless supplemented by iliac fixation, sacral screws by definition are the foundation of constructs that cross the lumbar-sacral junction.

Consequently, tremendous stresses are placed on these implants. Without the added strength of anterior cortical purchase, sacral screws, particularly in long constructs, are likely to fail. Lehman et al.,[45] using a cadaveric model, showed that placing the S1 screw into the sacral promontory ("tricortical" fixation) further increased the strength of fixation by 99%.

The feasibility of placing screws through the anterior cortex is based on the unique anatomy of the sacrum. Xu et al.[46] described the safe placement of screws into zone 1 (into the S1 body) and zone 2 (the sacral ala). Mirkovic et al.[47] describe medial (sacral body) and lateral (sacral ala) safe zones for purchase of screws with reduced risk of injuring vital anterior structures. Based on adult specimens, Mirkovic stated that the medial safe zone is located bilaterally 22 to 27 mm lateral to the midline of the S1 body. When the overhanging posterior ilium blocks the angling of the pedicle gear shift, the required 30 degrees from the midline a portion of the ilium may be removed without violating the sacral iliac joint.[46] Large screw diameters (7.5 mm and greater), particularly in male patients (males have a more narrow sacral width), make it more likely that the posterior iliac crest will interfere with screw placement.[48]

VERIFICATION OF SCREW PLACEMENT

In an effort to reduce the risk of injury to adjacent neural and vascular structures, intraoperative methods have been developed to verify screw position. During surgery, screw position can be checked by direct palpation, imaging, and neuro-monitoring.[49–51] The most common method used to check pedicle wall integrity is by manual exploration with a blunt-tip ball probe. Lehman et al.,[52] using a cadaveric model, demonstrated that even in experienced hands, a large percent of violations were not detected at surgery. The senior author, an experienced spine surgeon, failed to recognize 58% of medially violated pedicles and was unable to detect pedicle breach in 81% of cases. Probing, although still useful, is now combined with neuro-monitoring and/or radiographic verification.

Whitecloud et al.[53] demonstrated in a cadaveric study of spines instrumented from T12 to the sacrum that a true lateral image can fail to diagnose anterior cortical penetration. Even screws that appear to only cross 80% of the vertebral body (on a true lateral image) may have violated the anterior cortex. Violations became evident in 10% and 30% of cases in the low lumbar spine by rotating the gantry 5 and 10 degrees, respectively. All screws that cross the vertebral body by 50% or less did not violate the anterior cortex.

Kim et al.[51] describe a method that relies on a single posteroanterior (PA) image to assess screw placement as it relates to medial and lateral pedicle violation. This method was developed in an effort to reduce the radiation exposure for both the patient and the surgical team. Medial screw violation is suspected when the screw tip passes across the

midline of the vertebral body. Lateral violation is suspected when the screw does not cross the medial aspect of the pedicle. A high sensitivity has been reported with this method.

In addition to somatosensory-evoked potential (SSEP) and motor-evoked potential (MEP) that monitor the spinal cord's electrical conduction, electrophysiologists now use triggered electromyelography (EMG) to detect the presence of screws' contact with exiting nerve roots. These techniques have proven effective in both the lab and for the human lumbar[54–56] and thoracic spine.[55,57,58] Further discussion of these techniques is beyond the scope of this chapter.

SCREW PLACEMENT

Screws may be placed based on anatomic landmarks or with the assistance of fluoroscopic images. When pedicle screw placement is difficult secondary to pedicle size, rotation, or the absence of a cancellous center, a combination of techniques may be used (i.e., anatomic and fluoroscopic). Recent reports of increased radiation exposure to health care workers when using fluoroscopic techniques are concerning.[59] Radiation is increased by leaving one's hands in the field, standing on the projector side of the x-ray machine and when treating larger patients. Radiosensitive tissues (the eyes, thyroid, and gonads) may be at particular risk during cases when fluoroscopy is used to place a large number of screws.[60]

EXPOSURE

Using standard techniques, the patient is placed under a general anesthetic. At least 2 large-bore intravenous lines are placed. Neuromonitoring (triggered EMG, SSEP, and MEP) is routinely used unless there is a complete cervical spine injury. Monitoring of the upper extremity in patients with injuries at or below the thoracic spine can help prevent brachial plexopathies from arm position. A catheter is placed in the bladder. The patient is then log rolled into the prone position onto a radiolucent spinal table. All bony prominences are well padded, particularly over the ASIS. Many adolescents are thin, and skin breakdown can be problematic in this region. The abdomen hangs free to permit venous drainage. The knees are bent and the toes free from compression. The elbows are bent to 90 degrees and the shoulders abducted to less than 90 degrees.

The back is prepped and draped in the usually sterile fashion. A midline incision is placed over all instrumented levels. Radiographs are obtained prior to bony exposure to ensure that the arthrodesis does not extend outside the planned zone of fusion. The spinous processes are split longitudinally. Subperiosteal elevation from the spinous processes to the tips of the transverse processes is performed.

Care is taken avoid violation of the facet joints proximal and distal to the planned fusion.

LUMBAR SCREW PLACEMENT

The lumbar spine will be covered first because it is the region that most commonly receives screws and at least below the conus is the "safest" place to start placing screws early in the learning curve. Honing these skills initially in a cadaver laboratory is advised. The sacral and thoracic spine is discussed last. Screw placement is easiest to perform from caudal to cranial. The rationale behind this statement is that pedicles are larger distally (more margin for error) and therefore will help with orientation at the more proximal levels. Furthermore, low-lumbar screws are located in a "valley" and are more apt to become covered by blood later in the case. Anatomic landmarks are used to locate and place screws with laminotomy and imaging used as adjunctive techniques when screw placement is challenging.

The bony landmarks used in the lumbar spine are the transverse and mammary processes, the lateral aspect of the superior facet, and the lateral pars interarticularis (Fig. 56-6). At L4 the pedicle entrance is at the junction of a horizontal line along the middle of the transverse process and the superior facet. Cephalad to L4 the entrance becomes progressively superior to this line (for adults it lies above this line by 3.9 mm at L1, 2.8 mm at L2, and 1.4 mm at L3) and at L5 the entrance is inferior to this line.[61] The relationship of
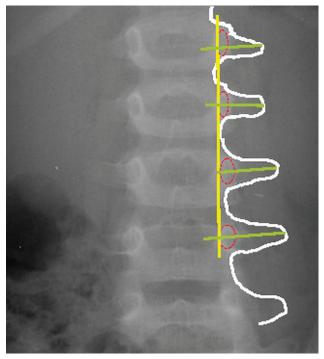

Fig. 56-6 AP radiograph demonstrating the relationship of the pedicles *(dotted ovals)* to the transverse processes.

the center of the transverse process to the pedicle can be seen on a standard PA radiograph at L1 through L4. At L5, lumbar lordosis alters the appearance of this relationship on a standard radiograph. The mammary process is located at the lateral borders of the pedicle. The pedicle entrance, which appears as a "blush" of bleeding cancellous bone, is exposed with a 4-mm high-speed bur.

The pedicle is then cannulated using a funnel technique. The gear shift should pass down the pedicle easily and with little pressure. Coronal orientation is approximately 30 degrees medial (posterior-lateral to anterior-medial) from vertical at L5 and decreasing to approximately 20 degrees at L4 with a 5-degree decrease at each level until L1. Sagittal plane orientation is achieved using two surface references (Fig. 56-7). The orientation is approximately 70 to 80 degrees from the pars interarticularis. Alternatively, the probe is placed perpendicular to a line connecting the edges of the segment's inferior lamina and the inferior lamina above. All screws are placed in a smooth line mindful of
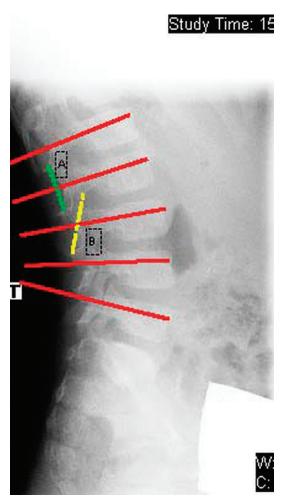

Fig. 56-7 Lateral radiograph of the lumbar spine demonstrating the sagittal plane trajectory of screws based on a path perpendicular to a line connecting adjacent laminas *(A)* or based on the laminar slope *(B)*.

maintaining a harmonious connection between screws in all planes. When there is a sharp deformity the perpendicular method is unreliable.

In the face of a spinal deformity screw placement becomes even more challenging. Several techniques can facilitate screw placement in these patients. The spinous processes are a helpful "guide" to the axial rotation of the spine. The spinous processes should not however be used as the sole guide to rotation when treating patients with severe juvenile or neuromuscular curves. With large curves the posterior elements may become dysmorphic; adjunctive techniques are therefore required. One can correct for axial rotation manually by placing the gear shift in a more horizontal position. Alternatively, the table can be rotated along the longitudinal axis of the patient to orient the segment into a more normal plane. The same technique works well for sagittal plane deformity, with the head of the bed raised or lowered as needed. If the table cannot rotate enough to allow for this more normal perspective, have your assistant press the flank in an anterior-medial direction from the convex side of the spine. The coupled motion that occurs with manual correction of the coronal deformity will derotate the spine and improve the orientation. When none of the previous techniques are working, a laminotomy and/or fluoroscopy is used.

When the probe is not passing easily, the problem is (1) gear-shift misdirection, (2) sclerotic pedicles (no cancellous core), or (3) extremely small pedicles. My preference when faced with either the (1) or (2) situation is to perform a laminotomy. The laminotomy is approached from the interlaminar space above (i.e., L2-L3 space to place the L3 pedicle). The portion of the spinous process that covers the interlaminar space is removed with a rongeur or bur. The superior edge of the inferior lamina is thinned with a bur or Kerrison rongeur. The midline junction of the right and left ligamentum flavum is thinned with a small rongeur until the epidural fat is visualized. A Woodson elevator is then placed under the ligamentum to clear the epidural vessels. Angled Kerrisons then resect the ligamentum from medial to lateral. The laminotomy is complete when the pedicle's medial, superior, and inferior borders can be palpated with a small Penfield. When necessary the slope of the pedicle can also be appreciated. Care is taken when palpating the pedicle because the traversing nerve root is just medial to the medial border of the pedicle. Bleeding when encountered is controlled with bipolar and/or Surgicel. Laminotomy also permits manual inspection of the outer pedicle cortex for breach after the screw is placed. If there has been a violation, threads are palpable outside the pedicle (unless the violation is lateral).

In the small or sclerotic pedicle, a gear shift may be too large and blunt to cannulate the pedicle. Under such a situation, a long smooth 0.62 wire may be gently drilled down the pedicle. Forcing a gear shift through bone increases the

risk of a visceral or neurovascular injury. The drill method is performed using high speed and a light "tap drill tap" technique. The "tap" is the palpation with the wire for a bony bottom. One should mark the wire 2 cm from the tip prior to drilling to avoid penetration beyond the anterior border of the vertebral body.

Doubt regarding the position of the wire is resolved by obtaining a fluoroscopic image. When there is a severe coronal deformity, one should place the fluoroscopic projector on the concave side of the patient to allow maximal rotation of the gantry. The C-arm is rotated in line with the pedicle. In this view, the pedicle demonstrates its maximum cranial-caudal and medial-lateral dimensions. The wire should enter the pedicle at the 3 o'clock or the 9 o'clock position for a left or right pedicle, respectively. The wire should be located within the pedicle lateral to the medial wall of the pedicle on the PA image and at the junction of the pedicle and body on the lateral image. If the wire misdirected, it should be repositioned at this point. Once satisfied with the wire's location, either a cannulated tap or a straight gear shift is placed down the pilot hole made by the wire. One should take care when using a cannulated tap. Wire advancement can occur. Lateral fluoroscopic images taken while tapping helps identify wire migration prior to anterior vertebral body breach. Wire advancement is more common when a "kink" is present in the wire.

Typically, the pedicle is tapped one size less than the intended screw unless the bone is particularly dense. In such a case, the pedicle should be tapped to the size of the screw. Maximum bony purchase is most important for short segment constructs. In these cases, the screw should cross approximately 80% of the vertebral body.

SACRAL SCREWS

The pedicles in the sacrum are the largest in the body and therefore the easiest to place. Screws at S1 can be placed either into the sacral prominence or the sacral alar (Figs. 56-8, 56-9, 56-10, and 56-11). Premonitory screws are biomechanically superior to alar screws and are generally the best choice. The pedicle entrance is at the lateral base of the S1 facet. A high-speed bur is helpful when removing the cortical bone. The sagittal orientation is 10 to 20 degrees cranial from perpendicular to the surface of the sacrum. The reason for this orientation is to maximize screw purchase by engaging the sacral prominence ("tricortical"). In the axial plane, the gear shift is aimed medially 30 degrees from the vertical. The iliac crest overhang may prevent optimal abduction of the probe. This can be addressed by either using a curved gear shift or making a small notch in the iliac wing. The gear shift is advanced to but not through the anterior cortex. The length of the hole is measured and 5 mm added for screw length. Several companies have adjustable punches that can be used to

open the anterior cortex without "plunging" through of the sacrum. Fluoroscopic images are valuable in checking screw position. Only a polyaxial screw can work in this location. When placing sacral screws it is important to remember that these implants tend to be prominent and may require removal.
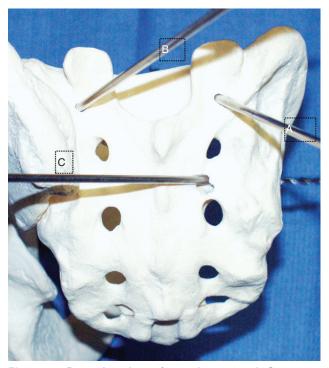

Fig. 56-8   Posterior view of sacral screws. *A,* S1 promontory. *B,* S1 alar. *C,* S2 alar.
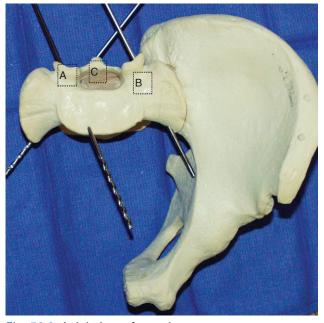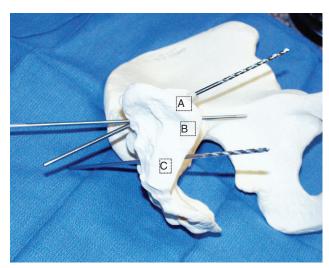

Fig. 56-9 Axial view of sacral screws.

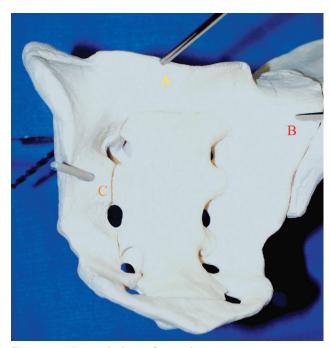Fig. 56-10 Anterior view of sacral screws.

Fig. 56-11 Lateral view of sacral screws.

The S1 alar screw is placed by opening a hole medial to the base of the S1 facet. The gear shift is place perpendicular to the sacrum in the sagittal plane and aimed laterally 40 degrees from the vertical in the axial plane. S2 screws are placed in the same manner. S2 screws are always alar. The entrance to S2 is just medial to a line connecting the S1 and S2 posterior foramina located 2/3 of the way down from S1. The screw is placed perpendicular to the sacrum in the sagittal plane and 45 degrees lateral in the axial plane.

THORACIC PEDICLE SCREWS

Fluoroscopically guided and free-hand techniques have been described to place thoracic pedicle screws.[28,62] Concern over radiation exposure has prompted many to seek a reliable method that minimizes this risk. The free-hand (aka funnel technique) relies on anatomic surface landmarks and a controlled manual decancellation of the center of the pedicle.[63] Excellent results have been reported using this method.[64]

As previously discussed, there are two pedicle screw trajectories, straight-ahead and anatomic. There are several advantages to the straight-ahead method. First it does not require a polyaxial screw. Monoaxial screws are stronger, are cheaper, and when used in coronal deformity produce greater correction. Second, straight-ahead does not violate the superior facet joint, an important concern at the cranial-end vertebrae. Lastly, straight-ahead is biomechanically stronger fixation than the anatomic method. Anatomic screw placement, however, still has value as a bailout should pedicle breach occur during screw preparation.

The entry point for the pedicle varies based on the level of thoracic vertebrae. At T10 to T12 the entrance can be found by merely removing the transverse process until it is flush with the lamina. From T4 to T9 the entrance is at the junction of the superior one third of the transverse process and the lateral one third of the superior facet. At T1 to T3 the entrance is at the lateral one third of the inferior facet and the midline of the transverse process. The entrance is visualized by removing the overlying cortical bone with a 4-mm bur. The inner cortical laminar bone is noted medial, sloping anteriorly. A curved probe with a 2-mm tip is gently placed through the pedicle with a light twisting motion. The probe is initially placed with its convex surface toward the canal (the tip is consequently aiming slightly lateral). After passing the 20-mm mark the probe is removed rotated 180 degrees, and advanced into the body. The angle of insertion is almost vertical at T12 and 5 to 10 degrees medial from T4 to T11 (Figs. 56-12, *A* and *B*). At T3 through T1 the angle gets progressively more medial with an angle of almost 25 degrees at T1.

The anatomic technique starts at the superior facet laminar junction. The pedicle probe is aimed 15 to 20 degrees inferiorly in the sagittal plane. As mentioned previously, a polyaxial screw is required when using an anatomic technique. As in the lumbar spine, laminotomy (Fig. 56-13) and/or fluoroscopy is useful when cannulating the pedicle is challenging (Fig. 56-14).

CONCLUSION

Children, like adults, have greatly benefited from advances in surgical technique. Although not the focus of this chapter, in this era of ballooning medical costs, it is no surprise that new technologies are more expensive than their older,
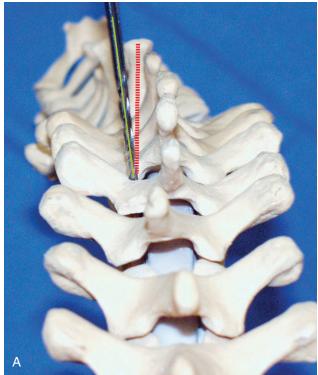
A

B

Fig. 56-12 *A,* Placement of thoracic screws using the ST technique. All screws are perpendicular to a line connecting the lamina. *B,* Axial orientation of screw placement.

Fig. 56-13 The image demonstrates palpation of the medial aspect of the pedicle following laminotomy.

more established counterparts (hooks and wires). However, there are times when the advantages of pedicle screw fixation make it the implant of choice. Screws are preferred when the posterior arch is absent and the spinal canal stenotic. Screws also may prove effective in limiting the zone of fusion and in avoiding the performance of a combined anterior/posterior procedure. The ability to safely and accurately place pedicle screws should be a skill mastered by all surgeons who treat the pediatric spinal patient.

A

Fig. 56-14 *A,* Preoperative radiograph of a severely deformed neuromuscular spine that required laminotomies and fluoroscopy to facilitate screw placement. *B,* Postoperative PA radiograph. *C* Postoperative lateral radiograph.
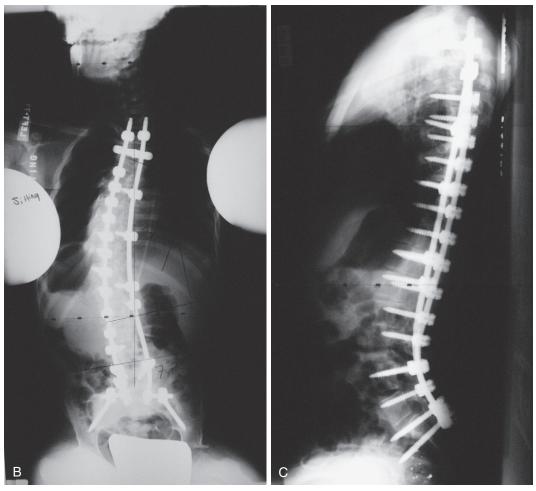
Fig. 56-14, Cont'd *B* and *C*

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Anterior Instrumentation Techniques for the Pediatric Spine

INTRODUCTION

In general, surgical indications for the treatment of pediatric spine trauma and associated instability are limited. This is especially true in the case of the pediatric cervical spine, which is largely due to the overall infrequency of pediatric spine injuries (<2% to 3% of all spine injuries), the increased mortality of these injuries, the improved prognosis for posterior capsuloligamentous and neuronal healing, and the small bony anatomy, which limits fixation potential.[1,2] Despite being the most commonly injured segment in the pediatric spine, most cervical spine injuries are treated non-operatively.[2] More specifically, the predilection for injury to the upper cervical spine stems from the inertia created by the size mismatch between the head and chest in children (especially those younger than 8 years).[3] Flexion distraction mechanisms predominately occur, which lead to posterior ligamentous distraction and anterior bony compression injury patterns.[4] Nevertheless, acute operative intervention for traumatic cervical spine lesions is rare and typically limited to posterior spinal fusion with or without instrumentation. Anterior approaches for spinal instrumentation are rarely indicated for pediatric cervical spine trauma.

The increased proclivity for anterior surgical fixation in the pediatric thoracolumbar and lumbar spine probably stems from a greater degree of comfort and familiarity with anterior instrumentation of these spinal segments. Additionally, the bony elements at these levels are large enough to support rigid internal fixation. Nevertheless, anterior spinal instrumentation for the treatment of spine trauma in pediatric patients at any spinal level has limited indications. This chapter is devoted to the surgical indications and techniques for anterior spinal instrumentation in pediatric spine trauma,

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which have been adapted almost exclusively from those used in the correction of pediatric deformity.

Anterior thoracolumbar spinal instrumentation has evolved from Dwyer's initial system in the 1960s to the rigid dual rod and plate systems available today.[5–8] Dwyer's system utilized a basic vertebral body screw, which remains an integral part of anterior spinal fixation. Conversely, the longitudinal member has undergone the most change over the past four decades, evolving from the flexible titanium cable used by Dwyer. The poor rigidity of the flexible titanium cable led to unacceptable rates of pseudarthrosis, kyphosis, and instrumentation failure. Following this, Zielke and others introduced a rigid anterior screw and rod system in the mid-1970s.[9,10] This system continued the use of a single bicortical vertebral body screw that was laterally placed approximately 20 degrees from posterior to anterior. The improved rigidity of the semirigid rod used in the Zielke system significantly reduced the rate of pseudarthrosis.[11,12] Over the next several years, Zielke's single-rod concept was improved on by several different surgeons, mostly by increasing the rigidity of the single rod.[13–15] Additionally, vertebral staples were introduced, thus improving screw pullout strengths by up to 50%.[16] The combined effect of these advancements was to improve fusion rates to those equivalent to instrumented posterior spinal fusion (82% to 100%)[14,17–20]

Synchronous to the advancement of single rod techniques, anterior plating systems were introduced.[21] These systems use rigid plates as longitudinal members and have experienced variable acceptance from spine surgeons. Although they are biomechanically equivalent to screw-rod systems, their fixed screw-hole position is less forgiving of anatomic variation, thus making placement of these implants technically more demanding. This increased difficulty stems from the fact that plating systems require the spine to adapt to the implant, whereas screw-rod systems adapt the implant to the spine. We do not use anterior spinal plating systems for this reason.

The next major advance came with the introduction of the dual rod concept. The Kaneda anterior Scoliosis System (KASS; Depuy Acromed, Rayhnam, MA) was originally designed for the surgical management of thoracolumbar fractures.[22] It improved on the perceived weaknesses of the single-rod systems, specifically: (1) weak implants, (2) suboptimal

sagittal plane correction, and (3) unacceptable correction losses at long-term follow-up.[7] This system included two flexible (4 mm) rods for improved stability while maintaining implant flexibility. Multiple dual-rod anterior systems are now available, each offering improved construct stability, elimination of postoperative bracing, and decreased pseudarthrosis rates[7,8,22–24] The demonstrated biomechanical and clinical advantage of dual-rod systems have made them a popular instrumentation system for anterior spinal surgery. Consequently, dual-rod systems are our instrumentation system of choice for pediatric and adult instrumented anterior spinal fusions.

INDICATIONS FOR ANTERIOR SPINAL FUSION IN PEDIATRIC SPINE TRAUMA

Indications for surgical fixation in pediatric spinel trauma follow the same general principles of adult spine trauma. Two absolute surgical indications are biomechanical instability and progressive neurologic deficit (Fig. 57-1). Fractures and/or dislocations that render the involved spinal motion segment unstable require operative intervention. Likewise, fractures with retropulsed fragments or disk herniations in which a progressive neurologic deficit is observed necessitate operative decompression and fusion. Outside of these absolute indications there are only relative indications. Because of the lack of controlled prospective studies evaluating the efficacy of surgical intervention for pediatric spinel trauma, the bulk of the support for these relative indications comes from small case series and clinical experience. Fractures that are not reducible by closed means or those in which reduction cannot be maintained by closed means are indicated for surgery. Pediatric dens fractures, the most common ($\geq$50%) cervical fracture in children, typically are close reducible and respond very well to halo immobilization.[1] However, some

Fig. 57-1 Photograph of left lateral decubitus positioning (right side up). Note right upper extremity in arm holder.

are irreducible or go onto to nonunion.[25] Both of these examples may require surgical intervention. Open fractures or those from penetrating trauma in which the projectile violates the transverse colon or the oropharyngeal space may require surgical removal of the fragment and débridement; however, current literature suggests that antibiotic therapy, while closely following serial C-reactive protein levels and radiographic imaging, may be sufficient.[26–29] Lastly, although acute operative indications for pediatric spinal trauma are uncommon, late surgical intervention for progressive traumatic kyphoscoliosis following spinal cord injury or postlaminectomy kyphosis is frequently required and is approached as an elective deformity correction case. Eighty-eight percent to 100% of patients who become tetraplegic prior to their pubertal growth spurt develop progressive spinal deformity with more than 60% requiring long posterior fusion, typically to the sacrum.[4,30–32]

One specific concept that merits further discussion is the influence of growth and development of the axial skeleton on surgical indications in pediatric spine trauma. The axial skeleton matures sooner in life than does the appendicular skeleton. As a result, fractures and/or dislocations that occur in adolescent patients can be evaluated and treated in a fashion similar to adult patients. On the other hand, spinel trauma in younger patients requires an individualized decision-making process and necessitates a basic understanding of the developmental anatomy of the spine. Similar to deformity correction surgery, an anterior approach may be most appropriate for prepubertal patients with significant anterior spinal growth remaining. We believe that patients with an open triradiate cartilage, Risser stage 0 or 1, premenarchal females, or those patients who have not yet reached their peak growth velocity should be treated with an anterior approach to prevent the crankshaft phenomenon.[33,34] Older patients or those with more severe injuries may be treated with posterior or combined approaches, respectively.

SURGICAL TECHNIQUE

CERVICAL SPINE

The most common surgical fixation in pediatric cervical spine trauma is the halo frame (see Chapter 51). Halo fixation in children has reported complication rates as high as 68%.[34] The most common complication is pin-site infection, especially in older children ($\geq$11 years). The anterior pin sites are most commonly involved. Most can be treated with local wound care and oral antibiotics (cephalexin or dicloxacillin). Pin loosening is another common problem. Despite these problems, halo fixation has been and continues to be the treatment of choice for most unstable cervical spine fractures in pediatric patients (Fig. 57-2).

Dens (C2) fractures account for 50% to 75% of pediatric cervical spine fractures, with most occurring as physeal injuries. The apophyseal growth plate of the dens closes around

Fig. 57-2 *A,B,* Standing AP and lateral full length radiographs of a 17-year-old girl who sustained a fall on her head while negotiating an obstacle course. Radiographs reveal a two-level compression fracture at T11 and T12. *C,* AP and lateral chest radiograph depicting sternum fracture, which is indicative of "four-column" injury and instability.

Continued

D

Fig. 57-2, cont'd *D*

age 6. Typically, dens fractures are closed reducible with mild cervical extension. Once reduced, these fractures can be treated in a halo vest for 6 to 12 weeks. Anterior instrumentation for dens fractures, although well described in the adult population, is very infrequently performed in pediatric patients.[25,35] Apfelbaum et al.[36] have previously described their technique for placing a direct anterior odontoid screw. Via a standard Smith-Robinson approach to the C5 vertebral body, a retropharyngeal plane, anterior to the C4 and C3 vertebral bodies is developed, thus exposing the inferior-anterior lip of C2, which is the entry point for the odontoid screw.[36] The guide, wire and cannulated screw are placed and oriented via fluoroscopic guidance.

Anterior instrumentation of subaxial spinal injuries is performed through a standard Smith-Robinson approach, as in the adult. This classic approach is well described in the literature and provides reliable access up to the body of C3. It uses the internervous plane between the medial edge of the sternocleidomastoid muscle and the anterior strap muscles. The common carotid artery, internal jugular vein, and vagus nerve are retracted within the carotid sheath laterally. Blunt dissection exposes the prevertebral fascia, which is sharply incised and the longus colli is subperiosteally elevated from a

midline to lateral direction to expose the vertebral body and disk space below. Placement of self-retaining retractors in this subperiosteal interval will protect the sympathetic chain that lies anteriorly along the longus colli muscle. Injury to the sympathic chain leads to Horner's syndrome.

Surgeon preference typically determines a left- versus right-side approach. Our preference is to approach from the left side, when possible. The anatomic course of the recurrent laryngeal nerve is more reliable on this side. Generally, it crosses from lateral, where it traverses under and around the aortic arch to lie between the esophagus and trachea below the C7 level.[37,38] Conversely, it traverses around the subclavian artery on the right side, which puts it at risk with more caudal (C6 and below) anterior cervical spine surgery. Nonetheless, the superiority of a left-sided approach has not been clinically proven, and many authors elect for a right, sided approach because it facilitates right-handed surgery. Regardless, careful attention to surgical and intubation technique is critical because Jung et al.[39] have demonstrated an overall incidence of 24.2% for laryngoscopy-proven vocal cord dysfunction following anterior cervical spine surgery. In two thirds of cases, this dysfunction was clinically silent; only 8.3% presented with clinical symptoms (hoarseness, cough).

Although recurrent laryngeal nerve injury leads to dysphonia and vocal cord dysfunction, the most common cause for postoperative airway issues following anterior cervical spine surgery is related to intubation techniques. Patients with cervical spine trauma present a set of challenges to the anesthesia provider because of the need for modified intubation techniques to the medical management issues related to neurogenic shock. Proper intubation techniques and patient positioning are, therefore, important considerations, especially in the pediatric patient. Children with spinal trauma must be immobilized at the injury scene and remain so until definitive fixation or orthosis application is complete. Pediatric patients have disproportionately large heads compared with their bodies, thus necessitating a cutout in the spine immobilization board or a pad for the trunk that allows the head to rest securely in a neutral flexion/extension posture. Pediatric patients placed on a flat (adult) backboard will lie in a position of cervical spine flexion. In the trauma patient, excellent and predictable results can be obtained with direct laryngoscopy and endotracheal intubation performed while a separate provider applies manual in-line stabilization (MILS).[40] This method is faster and more reliable than nasotracheal or awake fiberoptic intubation.[40] Further, nasotracheal intubation should not be used in patients with significant facial trauma. Although awake fiberoptic intubation has been shown to produce the least amount of cervical flexion and extension in cadaveric studies, patient cooperation may be challenging, if not impossible, in most pediatric patients.[41,42] Regardless, direct coordination with the anesthesia team is necessary to reduce the risk of iatrogenic injuries. Lastly, medial retraction against a firm endotracheal cuff may lead to recurrent laryngeal nerve (RLN) injury.

Consequently, some authorities have recommended monitoring cuff pressure and deflating and then re-inflating the cuff after retractor placement or repositioning.[43] This allows the cuff to re-center itself to reduce RLN compression and subsequent palsies. In young pediatric patients, cuffless endotracheal tubes are typically used, obviating the necessity of this maneuver.

THORACIC AND LUMBAR SPINE

The thoracic level is the least commonly injured level of the pediatric spine, because the ribs act as a fourth column of support. Like the cervical spine, most injuries to the thoracic spine are also typically treated nonoperatively. Even flexion-distraction injuries can be braced, typically in a thoracolumbar spinal orthosis (TLSO), and when surgery is needed it is performed posteriorly to restore the tension band. Nonetheless, surgery is indicated for instability or a progressive neurologic deficit. If there is no neurologic injury, then surgical indications are not absolute. When assessing stability, in the absence of pediatric specific biomechanical studies, we apply similar radiographic parameters as in the adult. Specifically, surgery is indicated for segmental kyphosis $\geq$25 to 30 degrees and/or loss $\geq$50% of anterior vertebral height. An anterior approach is indicated when canal decompression is required or when the approach may result in saving distal fusion levels (more commonly in cases of lower lumbar fractures). Given the limited indications, we rarely instrument the thoracic spine anteriorly for trauma.

As in adult trauma, the thoracolumbar and lumbar spine are the most frequently instrumented levels following pediatric spinal column injury. Anterior decompression and fusion is commonly used to treat mechanically unstable or neurologically progressive burst fractures (Fig. 57-3). Similar to the cervical spine, there are no clinical studies comparing nonoperative versus operative treatment of pediatric burst fractures. Likewise, there are no studies comparing anterior versus the different forms of posterior approaches. Instead, surgical indications are extrapolated from experience and literature for adult burst fractures.[21,44] Lalonde et al. reported a series of 11 pediatric burst fractures; five were treated with bracing and six with a posterior spinal fusion (PSF).[49] They concluded that nonoperative treatment was successful but resulted in a predictable mild progressive kyphosis (average, 9 degrees). On the other hand, PSF resulted in improved correction of anterior vertebral height with no loss of correction.

Although the posterior approach has several advantages, its main disadvantage is that it does not allow for direct decompression of the canal. In patients with a neurologic deficit, the anterior approach is often preferred because it allows direct decompression of the spinal canal.[22,24,45–47] The most important prognosticator of neurologic recovery is the severity of the deficit at presentation. Complete spinal cord injuries rarely demonstrate significant recovery, whereas incomplete injuries in children have an excellent potential for improvement.[48] Several clinical series have reported neurologic recovery following anterior decompression and fusion in patients with severe canal compromise and an incomplete neurologic deficit on presentation.[22,24,45,46,49,50] Similarly, McAfee et al.[45] reported that an anterior decompression and fusion in adult patients with an incomplete neurologic injury results in improvement of at least one motor grade in 88% of patients and return of bowel/bladder function in 37% of patients with conus medullaris syndrome. Thomas et al.[50] and McPhee[51] have reported similar success and improved outcomes in children following surgery.

The posterior approach primarily provides indirect decompression, which relies on ligamentotaxis to reduce the fracture and decompress the canal. This technique is not effective 2 to 3 weeks after injury because of early callus formation and healing. Alternatively, some direct decompression can be performed from a posterior approach. This requires laminotomy and retraction of the cord or thecal sac, followed by implosion of the retropulsed fracture fragments with a down-pushing curette. Although possible, this technique is challenging and still not as effective as anterior decompression. Posterior fusion in skeletally immature patients can result in the crankshaft phenomenon; however, the advent of pedicle screw (three-column) fixation and stronger implants has reduced this risk. Lastly, although short-segment posterior constructs are possible, in the setting of severe comminution and height loss, additional levels are required to achieve adequate stability. Because the anterior column is structurally restored during the anterior approach, only one level above and below the fracture must be fused, regardless of the severity of fracture. The anterior approach allows for placement of an anterior strut graft, which can incorporate, and thus off-load the instrumentation leading to a more permanent, stable spinal segment, with less propensity for late angular deformity.[45,46] With good surgical technique and rigid internal fixation, fusion rates of more than 90%, with minimal or no loss of correction over time, should be expected.[24,45,47,50] The following is a step-by-step description of the technique for performing corpectomy and anterior fusion in pediatric patients with thoracolumbar or lumbar burst fractures.

TECHNIQUE

SURGICAL APPROACH

The anterior thoracolumbar/retroperitoneal approach is the workhorse for anterior instrumentation in both trauma and deformity. Specifically, the retroperitoneal approach has been the standard since its introduction by Hodgson in the 1960s.[52,53] This approach provides excellent exposure for bony decompression, disk removal, structural grafting, instrumentation, and deformity correction in the injured thoracolumbar and lumbar spine. However, in pediatric
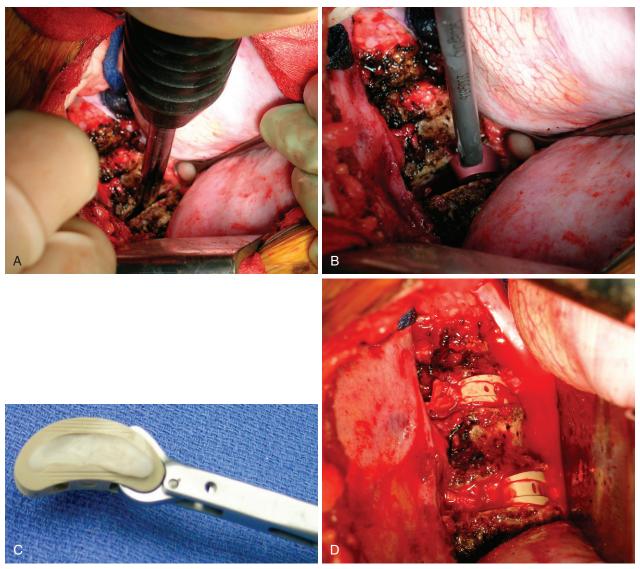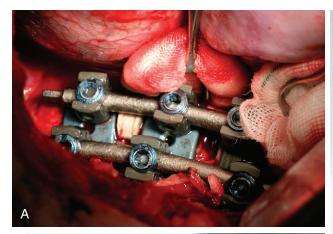
Fig. 57-3 *A,* Intraoperative photograph of T11-T12 diskectomy with Cobb elevator in disk space. *B,* Sizing of intervertebral spacer. Note positioning on anterior endplate. *C,* Poly-ether-ether-ketone (PEEK) spacer with absorbable collagen sponge (ACS) soaked with rhBMP-2 (Infuse, Medtronic Sofamor Danek, Memphis, TN). *D,* Intraoperative photograph of spacers in place.
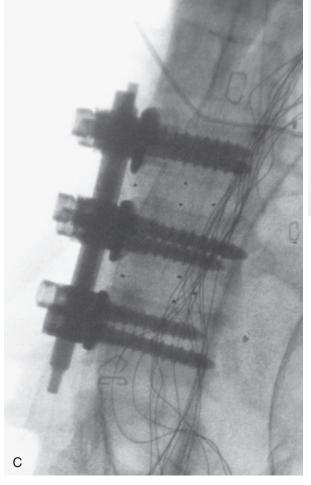
patients with spine trauma, the transperitoneal approach may also be considered. The main advantage of this approach would be in a patient undergoing laparotomy for an associated abdominal injury. Clean abdominal surgery (negative exploratory laparotomies) and thoracolumbar/lumbar spinal trauma could be approached through the same skin incision. Likewise, the transperitoneal approach should not be used in the setting of visceral perforation because the retroperitoneal plane, if preserved, would act as a barrier to contamination.

The thoracoabdominal approach compared with the posterior approach is typically less bloody because it traverses a well-defined avascular plane and does not require denervation of the paraspinal muscles.[54–56] We prefer to approach

from the left side (side of the thicker-walled aorta), with the patient placed in the right side–down decubitus position (Fig. 57-4).

The patient is placed onto an inflatable beanbag atop a radiolucent flat-top Jackson table (OSI, Union City, CA). Following this, the patient is log rolled into the lateral decubitus position, and an axillary roll is placed. The nondependent arm (left arm for a left thoracoabdominal approach) is placed across the body onto a well-arm holder or pillows. The hips and knees are flexed to relax the psoas muscle, and the bony prominences are padded. A pillow is placed between the knees. The shoulders and hips are then taped in place over a hand towel to securely immobilize the patient.
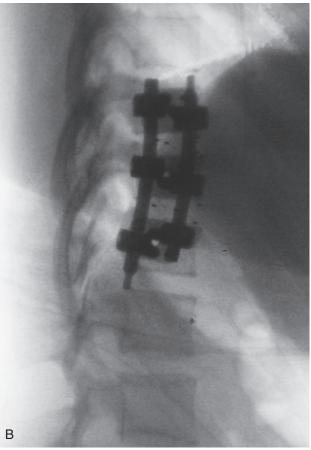
Fig. 57-4 *A,* Intraoperative photograph of dual-rod construct. *B, C,* Corresponding intraoperative AP and lateral fluoroscopic views.

The skin incision is usually made directly over the tenth rib.[55] The incision starts lateral to the paraspinal musculature and ends anteriorly, no further than the lateral edge of the rectus abdominis muscle. The incision is generally 10 to 12 cm, but it can be made expansile by carrying the incision caudally along the lateral border of the rectus sheath. The fascia and superficial muscles are divided in line with the incision. The rib is subperiosteally stripped, as attention is paid to avoid the neurovascular bundle on the caudal side of

the rib, and the rib is cut as far posteriorly as possible. This rib is saved on the back table and used for bone graft.

The chest cavity is then entered, and the lung is retracted or packed out of the field with a moist sponge; however lung retraction should be kept to a minimum, if possible. Next, the diaphragm is sharply incised approximately 1 to 2 cm from its attachment to the rib.[5,55,57] The remaining lumbar vertebrae are now exposed by developing a retroperitoneal plane with blunt dissection. Preoperative ureteral stenting

may be helpful when performing this less familiar approach in the setting of acute trauma. Intraoperatively, the stents are palpable and allow for safe and reliable identification of the ureters. We do not routinely use ureteral stents because they risk perioperative bacteremia.

VERTEBRAL EXPOSURE

Unlike deformity surgery, the involved vertebra(e) can usually be identified by direct visualization during surgery. Nevertheless, fluoroscopy can be used to confirm the exact vertebral level(s). Hematoma and any loose fragments should be evacuated. Copious irrigation helps to clear the view. The pleura over the thoracic vertebral bodies and the areolar tissue over the lumbar vertebral bodies are incised longitudinally in the midline and elevated from anterior to posterior, as a flap, to expose the lateral vertebral bodies. Care is taken when elevating this plane because the sympathetic chain lies within this tissue layer. The segmental arteries and veins are next dissected, ligated anteriorly, and tied with 2-0 silk

sutures on both ends. The segmental vessels should be ligated at the anterior or anterolateral margin of the vertebral body. Dissection and exposure of the involved level and approximately two thirds of the vertebral body above and below is all that is necessary.

Iliac and iliolumbar vessels will be encountered around the L4 level. The psoas muscle is elevated by blunt dissection to expose the lateral portions of the lumbar vertebral bodies. The sympathetic chain lies medial to the psoas muscle belly. Ogiela and Chan reported transient symptoms from sympathetic chain retractor neurapraxia in 100% of their patients, which resolved in all by 6 months.[11,57]

ANTERIOR CORPECTOMY—DIRECT CANAL DECOMPRESSION

Once exposed, the fractured vertebra is removed piecemeal. The disks above and below the injured body are removed as well (Fig. 57-5). McAfee et al.[45] advocate removal
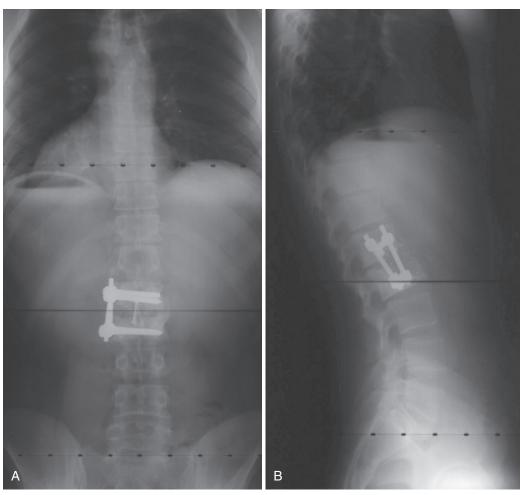

Fig. 57-5 *A, B,* AP and lateral postoperative radiographs at 1-year follow-up of hemicorpectomy with carbon fiber cage and dual-rod instrumentation/fusion of L1-L2. Note bridging trabecular bone. (DePuy Spine, Raynham, MA.)

of the posterior one half to one third of the vertebral body, leaving the anterior bony fragments in place as morcellized autograft. These authors recommend a very aggressive and wide decompression, including removal of the ipsilateral pedicle. Kaneda et al.[24] suggest that the adequacy of decompression and removal of the fractured vertebral body is confirmed by visualizing the contralateral pedicle. We follow a similar approach but do not routinely take down the ipsilateral pedicle. Regardless of surgeon-specific preferences, at the completion of the case the spinal canal must be fully decompressed. Thorough removal of all disk material is also essential to obtain a solid bony fusion. Li et al.[58] demonstrated that residual nucleus pulposus material in the disk space reduced the fusion rate from 70% to 20% in a porcine model. The anterior longitudinal ligament can be left intact to function as a tension band; however, we routinely remove it. In the case of a late presentation, the anterior longitudinal ligament may be contracted, thus requiring sectioning to allow for restoration of normal lumbar lordosis. Lastly, the remaining adjacent cartilaginous endplates are curetted to bleeding cancellous bone, without destroying the structural integrity of the endplate. In skeletally immature patients, the ring apophyses are resected completely.

VERTEBRAL SCREW AND ROD PLACEMENT

Once the fractured level has been prepared, a vertebral staple is placed on the lateral surface of the cephalad and caudad vertebrae. Staple placement is extremely important because the weakest link in anterior instrumentation systems is the bone-screw interface.[22,24,59] The KASS tetraspike staple (Depuy Acromed, Rayhnam, MA) increases axial screw pullout strength by 50%.[16] For dual-rod systems, it is important to place the staple in the mid-body, posterior enough to permit placement of the anterior screw. In the lower thoracic spine, the staple is placed just anterior to the rib head, or alternatively, a small amount of the rib head is removed with a rongeur to permit proper staple placement.[60]

Next the screw start point is prepared with an awl. The posterior screw is angled 10 to 20 degrees from posterior to anterior, and the anterior screw is placed transversely across the vertebral body. There are multiple vertebral staples currently on the market, and each has its own specifics regarding screw insertion and trajectory. Please consult the product manual for specific directions. All screws should be placed bicortical. A finger or periosteal elevator should be used to confirm that the screws breech the far cortex but are not prominent (<2 mm). As well, the posterior screw should not be completely seated on the vertebral staple prior to anterior screw placement because it will elevate the staple anteriorly off of the vertebral body. Both screws should parallel the vertebral endplates. Next, rods

are cut so that a minimum amount of rod (<3 mm) will protrude cephalad and caudad to the end screws. The posterior rod should be pre-bent to match the desired lumbar lordosis (Fig. 57-6).

BONE GRAFTING

The next step is bone grafting. The choice of graft is surgeon specific because multiple graft options have been described. Anterior tricortical iliac crest autograft has been recommended; however, harvest site pain and a large defect are significant drawbacks.[45,47] To reduce this, Behairy et al.[47] have described an alternative technique for harvesting the anterior iliac crest. They harvest a full-thickness (inner and outer table) window of bone, just inferior to the iliac crest. By leaving the crest intact, they maintain the normal contour of the iliac crest. In their series, all 20 patients fused and none had donor site-pain or complications at an average of 9-month follow-up. We have not had a similar favorable experience with anterior iliac crest harvest. Alternatively, rib, vertebral, and fibular autograft can be used.

Despite being considered the "gold standard" for bone graft, we feel the morbidity of iliac crest or fibular autograft strut harvest is too detrimental. Likewise, although resected rib is an excellent source for morcellized autograft, the thin, flat morphology of resected rib does not make it a good strut graft. For trauma, we commonly use morcellized vertebral body and resected rib autograft along with demineralized bone matrix to add bulk. This mix is packed into a structural mesh cylindrical metal cage.[61] Alternatively, strut autografts or allografts can be used without interbody cages or spacers. The most common allografts used are tibial, femoral, radial, or ulnar cortical struts.[12,62] Allografts can be picked to fit the anatomic gap, and they are easy to handle and fashion. The graft should occupy at least one half of the exposed endplates. It should be placed anteriorly along the strong outer rim of the endplate to minimize settling and facilitate restoration of vertebral height and lordosis. One or two struts can be used. Morcellized rib autograft is then placed to fill the remaining space, providing osteoinductive and osteogenic properties, not present in the allograft.

Developing a small trough in the adjacent endplates helps to prevent kickout of the graft or interbody cage but also weakens the endplate surfaces of the normal adjacent vertebral bodies.[45,63] This can lead to subsidence and kyphosis. To avoid this potential complication, the graft or interbody mesh cage should be placed "press fit" to the endplates. Prior to placing the graft or cage, the interbody space is distracted with a distraction device affixed to the screw heads. Alternatively, a fist can be placed into the posterior midline to wedge open the space anteriorly. Once the graft or cage is in place, the distraction is released and

Fig. 57-6 *A-B,* Photographs of molded thoracolumbar spinal orthosis (TLSO).

the pre-bent posterior rod is placed. Segmental compression is then applied to improve sagittal contour, enhance fusion rate, and reduce the risk of graft kick out. Once the posterior rod is secure, the anterior rod is placed to reinforce the construct.

CASE COMPLETION AND CLOSURE

Intraoperative anteroposterior (AP) and lateral radiographs are obtained to confirm coronal and sagittal alignment. All set screws are checked and retightened. A cross-link may be desired to increase torsional rigidity, but this usually is not necessary. The pleura is closed with 2-0 chromic suture, and a chest tube is placed if the thoracic cavity was entered. The diaphragm is repaired with interrupted 2-0 silk sutures, whereas the internal oblique and transversalis muscle layers can be closed in an interrupted or continuous fashion. Our preference is for interrupted 0 Vicryl sutures. The external oblique muscle is repaired in a similar manner. Care should be taken to avoid the intercostal nerve to

prevent post-thoracotomy syndrome. Absorbable sutures are used for the subcutaneous layers and Steri-strips are placed prior to sterile dressing sponges.

POSTOPERATIVE MANAGEMENT AND BRACING

Patients are allowed to bear weight and ambulate on the first postoperative day. In the absence of other associated medical/surgical problems related to their trauma, patients are discharged when they are able to ambulate at least 100 feet with a walker, ascend/descend stairs and pain is well controlled. Physical therapy is not necessary but can be helpful. Patients should be encouraged to walk as much as they feel comfortable with for the first month. Sports are high demand activities and are not reintroduced until a solid fusion has been achieved. In the presence of a solid fusion, patients are allowed to return to their previous activity and sports.

Standing PA and lateral radiographs are obtained prior to discharge, and at 6 weeks, 3 months, 6 months and 1-year postoperative. They are also obtained at 2 and 5 years to

evaluate for fusion, implant failure, decompensation, and curve progression.

TLSO or casts have been recommended by some surgeons following anterior spinal fusion in adolescents for 3 to 6 months or until fusion is evident.[17,57] More recently, improved instrumentation systems have negated the need for postoperative bracing following anterior spinal fusion for deformity correction; however, because of reduced stability of anterior instrumented constructs compared with the native vertebral body, most spine surgeons, would agree TLSO bracing is warranted following anterior spinal fusion for pediatric spinal trauma. Smaller patients treated with dual-rod systems and metallic interbody cage supports may not require TLSO bracing. Regardless of the clinical importance of the construct's biomechanical stability, a TLSO brace also serves an as important reminder to younger patients and their parents that activities need to be significantly curtailed until the postoperative healing phase is complete (12 to 24 weeks) (Fig. 57-7).

CONCLUSION

The overwhelming majority of pediatric spine trauma patient can and should be treated nonoperatively. Rare cases in which closed reduction is not possible or more frequently when neurologic deficits exist at presentation, surgical intervention may be warranted. The two absolute indications for surgical intervention of pediatric spine fractures are (1) biomechanical instability and (2) progressive neurologic deficit. The most common pediatric spine fracture treated via anterior instrumentation is the unstable burst fracture. Corpectomy and instrumented anterior spinal fusion using a dual-rod system with titanium mesh cages or allograft struts provide excellent radiographic results with reliable improvement in neurologic status, especially in patients with incomplete injuries.

ACKNOWLEDGMENT
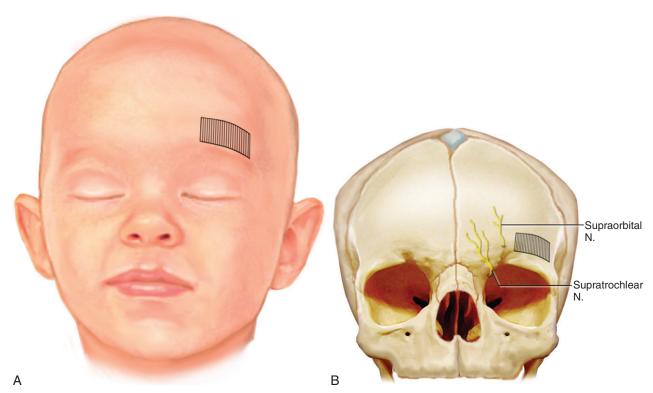
Fig. 57-7 *A-B,* Oblique and AP schematic of face/skull depicting safe zone for halo-ring pins superior to the outer aspect of the eyebrow *(hashed area).* Note location of supraorbital and supratrochlear nerves over orbital socket. Optimal halo-pin number is generally considered to be 10 to 12 pins at 1 to 2 in-lb. torque in small children, compared with 4 pins at 8 in-lb. torque in adults.

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C H A P T E R

58

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CHRISTOPHER M. BONO

Gunshot Wounds to the Spine

INTRODUCTION

A report of traumatic injury to the spine dates back to 1700 BC.[1] Galen, who is considered a pioneer of spinal surgery (130 to 200 AD), described that longitudinal lesions to the cord did not cause severe functional damage, whereas transverse lesions were associated with paraplegia below the lesion. He also described the relation between the level of the lesion and neurologic symptoms.[2] Decompressive laminectomies for the treatment of acute spinal compressive lesions were first recommended by Paul (625 to 690 AD).[3] A bullet injury to the lumbar vertebra was described by Geraud in the year 1753. In the American Civil War, hundreds of cases of gunshot wounds to the spine occurred and death rates were as high as 55%.[4] During World War I (1914 to 1918), the rate of death resulting from gunshot wounds to the spine was more than 60%,[5] possibly because of the use of more powerful weapons. During World War II (1939 to 1945), the rate of death resulting from gunshot wounds to the spine was markedly reduced to 7.4% to 14.5%, which has been attributed to the use of antibiotics and improved trauma resuscitation techniques.[5–7] During the Korean War (1950 to 1953), all penetrating spinal injuries underwent surgical exploration and some surgeons even reported significant neurologic improvements.[8]

The number of civilian penetrating spinal injuries has consistently increased during the last 3 decades, and the majority are caused by gunshot.[9] In one large study, gunshot wounds were the most common cause for thoracic spinal injuries.[10]

EPIDEMIOLOGY

The number of gunshot injuries throughout the world has continuously risen.[11] Penetrating injuries to the spine are a major cause of spinal cord injuries in the United States. Of 6014 cases reported to the National Spinal Cord Injury Data Research Center during an 8-year period, 13% were the result of gunshot wounds, the second leading cause after automobile accidents.[12,13] Gunshot wounds in civilians often are caused by low-velocity, smaller caliber guns.[14] Guns with calibers of .22, .25, .32, and .38 are the most commonly used weapons, and young males are the most frequent targets.[15,16] According to a study conducted in the United States, African Americans receive 53% of gunshot wounds, Latinos receive 28%, Caucasians receive 18%, and Asians receive 1%. The most commonly affected area was the thoracic region, and the next most common were the lumbosacral and cervical regions.[17] Spinal cord injuries resulting from gunshots are more likely to result in complete sensorimotor paralysis compared with spinal cord injuries resulting from blunt trauma. Approximately 50% of spinal cord injuries resulting from gunshot wounds affect the lower extremities and 50% affect all four limbs. Incomplete injuries have a much better prognosis.[18]

Injuries to the spinal cord and/or vertebral column are relatively uncommon in the pediatric population. With an incidence ranging from 1% to 10% of all spinal injuries, approximately 4% are caused by gunshots.[19] The injury patterns observed in young children differ from those in adolescents and adults, largely because of age-dependent variations in the anatomic and biomechanical features of the spine. Children also have a higher incidence of complete spinal cord injury and spinal cord injury without radiographic abnormality (SCIWORA). Pediatric spinal injuries can present special problems involving external spinal immobilization and surgical intervention in a child with significant growth potential.[19]

BALLISTICS AND BULLET DESIGN

To understand the mechanism of gunshot injuries, it is important to understand the nature of firearms and their projectiles. Ballistics is defined as the scientific study of projectile motion and is divided into three categories: internal, external, and terminal ballistics. Internal ballistics has to do with the projectile within the firearm. External ballistics has to do with the projectile in the air. Terminal ballistics has to do with what happens when the projectile hits its target. Wound

ballistics is a subset of terminal ballistics and is the most important aspect of ballistics for physicians to understand. However, a basic understanding of all aspects of ballistics is helpful in understanding the wounding process.

The destructive power of the bullet is determined by its mass and speed, and an increase in velocity will have an exponential effect on the energy of the injury. Muzzle velocities between 1000 and 2000 ft/sec are considered low energy, whereas those above 2000 ft/sec are considered high energy. Low-velocity, low-energy firearms include pistols and handguns; high-velocity, high-energy weapons include military assault rifles.[18]

Before firing, the lead bullet is held firmly in the end of a brass cartridge case. This cartridge case contains a flammable propellant, known as the charge, and has a primer at its base. When the firing pin of the gun strikes the primer, the primer is detonated, which ignites the charge within the cartridge case. The burning gases expand and propel the bullet from the cartridge case and along the barrel of the gun. Spiral grooves within the barrel of the gun, known as rifling, grip the bullet and cause it to spin around its long axis. This spinning creates a gyroscopic effect, that provides the bullet with directional stability in the air, enabling it to travel straighter and be more accurate than a nonspinning projectile. The longer the barrel is, the more time the bullet has to accelerate and the faster it will be going when it leaves the gun. Because rifles have longer barrels than handguns, rifle bullets leave the gun with much higher velocities than do handgun bullets.[20,21] Shotgun wounds are more complicated because of the combined mass of the numerous fired pellets and the large pieces of "wadding" that accompany the metallic fragments.[18]

The type of injury in the civilian population is mainly low energy, but the scenario is changing because of the availability of high-energy firearms to civilians. An increasing number of high-energy injuries are being reported. Previously, high-energy injuries occurred mainly in the military population.[22] It is important to differentiate between high- and low-energy gunshot injuries because the difference plays a crucial role in treatment.

Bullet energy is not the only determinant of the extent of injury. Local tissue factors and the distance (range) between the weapon and the victim both have a major effect on these injuries. It is, therefore, important to be aware of the way projectiles behave before and after impact and also of the manner in which various tissues respond to these injuries. Fragmentation of the missile can increase the zone of tissue-destruction. Hollow point bullets explode on impact, which leads to multiple fragmentation and deviation from the linear path. The phenomenon of yaw refers to the tumbling of a bullet along the longitudinal axis. A longer bullet has a greater yaw and circumference of destruction. Bullets can be nonjacketed, partially jacketed, or fully jacketed. Fully jacketed bullets exhibit little deformation with firing and are intended for long-range attacks. They are very precise and

cause clean entry and exit wounds. The other two types expand on impact and exponentially expand the circumference of tissue damage.[18]

Many different materials have been used for bullet production. Most of them are made of a lead core, which might be combined with different metals to achieve the desired hardness. These substances can have both systemic and local toxicities on tissues. Lead poisoning has been reported with bullets lodged in the intervertebral disk, although it is more common in the synovial joints of the extremities.[23] Although cerebrospinal fluid (CSF) would intuitively be an effective solvent, systemic lead elution with bullets located in the intramedullary substance of the spinal cord can occur. In the case of a retained lead bullet, a significant increase in serum lead levels and characteristic hematopoietic changes can be an indication for bullet removal. However, in actual practice, bullets very rarely need to be removed because of lead toxicity.

Some authors recommend removal of copper-containing bullets from the spinal canal.[24,25] In vivo experiments in monkeys have shown the local necrotic effect of copper on brain tissue.[26] Nevertheless, in many instances, the material composition of the bullet is not known.

CLINICAL MANAGEMENT

The initial focus of the gunshot patient must address all life-threatening injuries. Maintenance of airway, breathing, and circulation (ABC) are the most important factors. The evaluation of the patient with a gunshot wound to the spine begins with a history and a thorough physical examination. The history might be difficult to obtain in many settings of gun violence, but, when possible, knowledge of the weapon involved, bullet, and trajectory of the injury can be helpful. Such knowledge can provide clues to the injury pattern and potential secondary effects of the line of penetration of the projectile. If the patient is conscious and stable, a neurologic evaluation should be obtained to determine any numbness or weakness the patient perceives.

The keys to the physical examination are an assessment of the entrance and exit wounds and a complete neurologic examination. The entrance and exit wounds will provide clues to the path of the bullet before it encountered the spine. This is particularly important if the possibility of bowel penetration and contamination is present. The neurologic level of injury is defined as the most caudal segment that tests as intact for sensory and motor function on both sides of the body. By convention, to be considered motor intact, a muscle must be graded at least fair (3 on a 5-point scale). The motor level is then defined as the level of the lowest key muscle having a grade of at least fair. All levels above this must be intact.[27] The neurologic evaluation must be complete to include motor function, sensation, reflexes, and rectal examination. The examination can be tailored to the clinical presentation. For instance, no upper extremity deficit would be

expected with a gunshot wound to the lumbar spine. The entire examination should be recorded meticulously because changes in this examination with time might have treatment implications.[13]

Particular consideration should be given to the region of injury. Gunshot wounds to the neck often are complicated by tracheal injuries, which might require emergency intubation or tracheostomy.[28] The radiologic evaluation should be conducted as soon as possible, but the majority of fractures are inherently stable.[18] In cases of pulsatile neck swelling, vascular damage should be suspected. The most commonly affected arteries are the carotid and vertebral arteries. If detected, an emergency vascular repair is recommended to restore cerebral blood flow. Other wounds in the neck region must be detected and evaluated because pharyngeal and esophageal wounds often are associated with higher rates of infections. Endoscopic techniques have been used effectively for surveillance in the acute setting.[28]

Unique features of the pediatric spine create differences in the evaluation of neck injuries in children. For example, the fulcrum of movement is higher because of a proportionally larger head size, the vertebrae might be incompletely ossified, the ligamentous attachments might be stronger than bone, and the articular surfaces are more horizontal. In addition, significant spinal cord injury can occur without radiologic abnormality in children, especially those younger than 8 years.[29,30]

The region of the spine most commonly affected by gunshot wounds is the thoracic region.[10] Vital organs, such as the heart, lungs, and great vessels, are at risk of injuries and ventilation and hemodynamic stabilization should be the primary goal. A detailed chest and cardiac evaluation is vital for the detection of pneumothorax, hemothorax, cardiac perforation, tamponade, and aortic disruption. Abdominal examination is another important aspect because injury to the organs in the abdomen can be fatal if not detected early. Colonic perforation leading to wound contamination has a high rate of spinal infection, which can occur up to 6 months after the initial insult.[31] Sacral gunshot wounds are mainly complicated by hemorrhage. Sterile packing of the wound with methyl cellulose and bone wax has been used and is an effective method of hemostasis. Emergency angiographic embolization can stop the uncontrolled bleeding, but it might cause devascularization of vital neural structures.[32]

SPECIFIC EVALUATION OF SPINAL INJURY

Once the trauma team has stabilized the patient, the focus is turned toward the spinal injury. When available, witnesses, emergency medical technicians, and law enforcement officers can provide useful information regarding the direction of the injury, the type of weapon or bullet, and the proximity of the shot. Often, a conscious patient can account for the number

of shots fired. The need for immediate cervical stabilization with a collar usually is not necessary.

The patient must be stripped of all clothing. Entrance and exit wounds should be examined, and palpation of the tissue is performed to assess the presence of crepitation and the general turgor of the tissue. A very large exit wound with crepitus and increased tissue turgor is consistent with wounds that have a large permanent cavity and might very well have significant tissue necrosis.[33] Often, however, only an entrance hole can be found because the bullet is lodged in tissue. The entrance wound can be identified by its characteristic well-defined and cleaner margins. The exit wound has more of a "blown out" appearance because of the phenomenon of yaw and fragmentation of the bullet. In the abdomen, it is critical to determine the path traveled by the bullet. Although deep probing is not recommended, superficial inspection for imbedded pieces of clothing or foreign materials is useful. A culture specimen from the wound track can be obtained, but this practice is not well supported in the literature. Local wound care and radiopaque markers (e.g., electrocardiogram leads) are placed over all wounds to facilitate radiographic deduction of the gunshot path.

The physical examination should continue as for all other spinal trauma. All spinous processes must be palpated for tenderness and crepitus. An in-depth neurologic examination, including motor, sensory, reflexes, and anal sphincter tone, is mandatory and must be documented precisely. This can sometimes be difficult with pediatric patients because they are more agitated and uncooperative. In the intubated or unconscious patient, neurologic testing is limited. However, if paralytic agents have not been administered, deep tendon and bulbocavernosus reflexes can still be elicited.

Traditionally, general surgeons have strongly advised in favor of operative exploration of neck wounds. Current recommendations advocate that the wound should be explored only in patients with specific warning signs of serious injury. Otherwise, they should only be observed.[34]

RADIOGRAPHIC IMAGING AND DIAGNOSTIC TESTS

Imaging and other investigations are the same in adults and children but the anatomic differences should be kept in mind. Standard anteroposterior and lateral view plain radiographs of the spine usually can locate fractures and bullet fragments (Fig. 58-1 A and B). With the help of the bullet markers, the bullet path of the projectile can be traced. The location of the bullet can be in soft tissue, bone, intervertebral disk space, or within the spinal canal itself. Vertebral body height, interpedicular distance, and adjacent segment kyphosis should be measured. Gunshot wounds rarely cause unstable spine fractures.[24] In neurologically intact and fully conscious patients, dynamic flexion and extension views of the cervical spine are of great help in patients with questionable spinal stability.
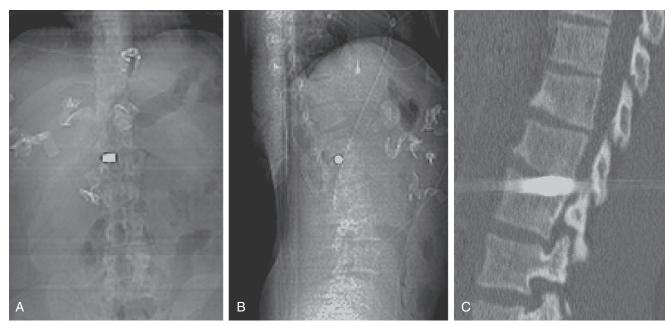
Fig. 58-1 Anteroposterior *(A)* and lateral *(B)* view plain radiographs. This 25-year-old male patient was shot in the chest with the bullet entering the spine from the left lateral side. The patient had no neurologic compromise, and no neurosurgical interventions were undertaken. The patient was discharged home having achieved full recovery. *C,* Sagittal view CT scan shows the bullet fragment in the posterior aspect of the T12-L1 interspace without any evidence of fracture.

One study showed that wounds limited to the calvaria do not cause trauma to the cervical spine.[35] Kihtir et al.[36] found that spinal fractures were noted in 10% and 20% of midface (maxillary) and orbital (frontal) injuries, respectively, whereas none are noted after gunshots to the lower face (mandibular region). As a result, gunshot wounds to the maxillary and orbitofrontal region should be investigated more aggressively.

Helical computed tomography (CT) has gained wide acceptance in the evaluation of a variety of traumatic and nontraumatic emergency conditions. High-quality diagnostic images are obtained in a short time, which is an essential factor to be considered for critically ill patients. It also is more comfortable for the patient. In the emergency setting, helical CT has gradually replaced traditional imaging techniques such as conventional radiography, conventional contrast-enhanced studies, and conventional angiography. Transverse images usually are sufficient to make a proper diagnosis in most cases. Reformatted and three-dimensional images are complementary in complex cases (Figs. 58-1, *C* and 58-2). These images also are useful in planning the surgical procedure, because the surgeon usually prefers the reconstructions that more closely resemble the conventional angiograms.[37] Currently, this is the modality of choice for spinal gunshot trauma.[38] The limitations include artifacts caused by metal, such as dental fillings or bullet fragments, which can obscure the detail visualization.

The use of magnetic resonance imaging (MRI) to evaluate gunshots of the spine is controversial. Bullet migration from the pull of the strong magnet is a relevant issue and can possibly lead to further neurologic or soft tissue damage. This issue remains theoretical in that numerous reports of MRI after gunshot wounds to the spine have not supported this concern.[18] Another disadvantage is the problem of compatibility of the support equipment, which might not be compatible with the magnet.[37] The advantages of MRI over CT include markedly less artifact, better soft tissue imaging, and coronal, sagittal, and axial visualization of the neural elements.[39] Although these advantages are attractive, the use of MRI should be based on the patient's specific needs. In our experience, the most frequent complaint with MRI is that the patient might feel a sensation of heat from the bullet, which can cause the study to be aborted. This is particularly common with jacketed bullets.

Other diagnostics, such as color Doppler ultrasonography, have been proposed as noninvasive tests for the diagnosis of traumatic vascular injuries. Color Doppler ultrasonography provides adequate information regarding flow, lumen, vascular wall, and thrombus features.[40] Color Doppler ultrasonography performed in patients with penetrating trauma to the neck has shown promising study results.[41]

TREATMENT

The treatment of spinal injury from gunshot wounds should be individualized based on the age of the patient, level and type of injury, extent of neurologic dysfunction, and presence of other associated injuries. Most of the basic principles applied in treating adult patients with spinal cord injury also are applicable to the pediatric patients.
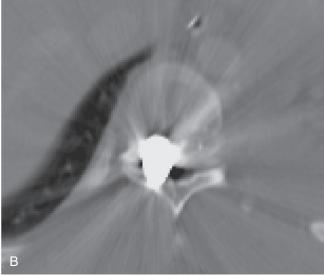
Fig. 58-2 Axial view CT scans *(A* and *B)* show the bullet in the spinal canal at the T10-T11 level in a 35-year-old male patient who sustained a single gunshot wound.

Once the ABCs of the patient have been stabilized, management can be started by using a structured algorithm. One study has noted significantly higher numbers of mortalities for cases in which the treatment deviated from the algorithm.[10] Although such programmed treatment protocol has obvious advantages, it has shown only minimal impact on outcome.

PROPHYLAXIS AND ANTIBIOSIS

Tetanus prophylaxis must be considered in all instances of spinal gunshot wounds. If any question remains regarding the patient's most recent immunization, the victim should receive tetanus prophylaxis in the emergency department at the time of the initial evaluation.

Broad-spectrum antibiosis, based on the region of injury and local hospital bacterial sensitivity, should be initiated immediately. It has been shown that the heat generated by the firing of the bullet does not autosterilize it.[42] In addition, bullet entry has a vacuum effect in which outside air and contents can be pulled into the wound, thus contaminating it. Recommendations for the duration of antibiosis vary. For gunshot wounds not complicated by viscus perforations, it generally is recommended to maintain 48 to 72 hours of prophylaxis. However, decisions must be made considering the clinical evidence of infection both in the spinal and extraspinal regions.

The risk of infection always is greater in patients with viscus perforation.[18] The infection rates are highest with colonic wounds that occur before the bullet enters the spine.[43] Kitchel[13] suggests that high-velocity bullets cause more tissue destruction and carry higher risk of wound contamination and recommends débridement and fragment removal with parenteral antibiotics when viscus perforation has occurred. Alternatively, Heary et al.[32] reported that bullet removal and

surgical débridement have been associated with six-fold higher rates of infection compared with antibiotics alone. Antibiotic therapy for 7 to 14 days has been recommended by many authors, with the lowest infection rates occurring in patients with colonic perforation.[31,43] Except for indications such as neurologic deterioration or lead toxicity, bullet extraction is not advocated to decrease the infection risk.[44]

The role of antibiotic prophylaxis after esophageal and upper airway perforation is less clear. Pooled secretions in the hypopharynx are thought to increase the severity of infection with gunshot wounds to this area.[28] The decision to explore these wounds usually is based on the size of the lesion; smaller wounds can effectively be treated nonoperatively. With clear evidence of frank infection or meningitis, it is advocated to extend prophylaxis for 7 days. To our knowledge, no controlled study of the duration of the antibiotic prophylaxis after gunshot wounds has been conducted.

Pain should be managed aggressively. Occasional use of neuraxial analgesics, after assessing the type of injury and hemodynamic stability, has been advocated.[13]

ASSOCIATED INJURIES

During the initial treatment of patients with gunshot wounds, the bullet entry site should be treated with débridement of any devitalized skin and superficial soft tissues. If a CSF leak seems to be present, a subarachnoid drain can be placed. In cases in which a persistent CSF leak or fistula is present through the bullet entry or exit sites at the level of the skin, open surgery must be considered.[18] A fistulous connection with the bowel, bladder, or pleural cavity might also be present and should be treated with surgical exploration.[45] Because of the risk of meningitis resulting from a persistent CSF leak, the treatment should involve a laminectomy with

repair of the dural violation, either primarily or with use of a dural graft.[46,47] In these relatively rare instances, placement of a temporary lumbar subarachnoid drain after laminectomy might be beneficial to supplement the dural repair.

PATIENTS WITH NO NEUROLOGIC DEFICIT

The treatment of gunshot wounds to the spine of a neurologically intact patient is similar to the treatment of blunt trauma. In the neurologically intact patient, spinal stability must be assessed. Almost 10% of patients with gunshot wounds sustain spine injury. Two thirds of the spine injuries are significant, and approximately 13% are unsuspected.[48]

Plain radiography and CT are helpful. The three-column theory presented by Denis,[49] which was designated for blunt trauma only, does not apply to penetrating trauma. In contrast to blunt trauma, two- or even three-column disruption is less likely to result in instability for cases of gunshot wounds. In a study conducted by Denis, the proposed mechanism of injury implied an abrupt acceleration/deceleration of the body/spine in space. With gunshot wounds, the body/spine can be considered stationary and the bullet is the directional force. In the best case scenario, a through-and-through bullet wound will damage only those structures that lie directly in its effective path. Low-energy gunshots have a narrower circumference of damage than do high-energy wounds. These factors influence the amount of spinal instability after gunshots to the spine.

As mentioned earlier, in conscious, cooperative, and neurologically intact patients, dynamic lateral view radiographs should be obtained. Careful flexion and extension of the spine can reveal pathologic mobility of the adjacent spinal segments. In the cervical spine, commonly used criteria for radiographic instability are greater than 11 degrees of angulatory change or more than 3.5 mm of translation between flexion and extension views. In the L5-S1 segment, more than 5 mm of translation or 15 degrees of angulation is considered unstable.[50] If instability is detected, the affected segment can be fused with instrumentation.

Common indications for decompression in neurologically intact patients are lead poisoning from bullet and contact of a copper-jacketed missile with the neural elements.[18] If the fragment is located in the joint space, an increased risk of lead intoxication is present and symptoms can occur years after the injury.[45] If surgery is planned to retrieve the fragment, chelation therapy is recommended before surgery. Routine removal of the bullet is not advised and is associated with a high rate of complications.

PATIENTS WITH NEUROLOGIC DEFICIT

Gunshot wounds commonly result in complete spinal cord injuries, which might be paraplegia or tetraplegia depending on the level of injury (Fig. 58-3). Incomplete injury can present in a variety of manners, including Brown-Sequard, central cord, anterior cord, and, in rare cases, cruciate hemiplegic
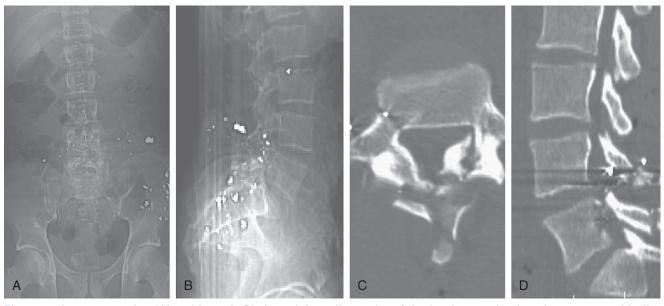

Fig. 58-3 Anteroposterior *(A)* and lateral *(B)* view plain radiographs of the lumbosacral spine show scattered bullet fragments and fracture of L4-L5 in a 26-year-old-male patient who presented with gunshot wounds to the abdomen and back. The patient had 0/5 strength in the lower extremities from mid calf to foot bilaterally, with decreased sensation. Axial *(C)* and sagittal *(D)* view CT scans show multiple fractures of L4-L5 with bony and bullet fragments in the spinal canal. The patient underwent L4-L5 laminectomy and L3 to S1 posterior instrumentation and fusion after 4 days. The neurologic status remained unchanged at the time of discharge.

syndromes.[39] This depends on the level and severity of injury (Fig. 58-4).

The role of corticosteroids in the treatment of gunshot injury has been questioned. Some studies have shown that patients treated with methylprednisolone or dexamethasone, compared with those treated without it, have a higher incidence of complications in the patients who received steroids. No difference in outcome of neurologic function was shown when using the American Spinal Injury Association motor score. Other complications, such as infection and gastrointestinal complications, also were reported to be higher in the steroid group.[32,51,52] These data suggest that corticosteroid medications do not play a role in spinal cord injuries caused by gunshot wounds.

The role of surgery in gunshot injuries to the spine also is limited. Generally, it does not improve neurologic symptoms. Surgical decompression of cervical and thoracic gunshot wounds has no beneficial effect on the likelihood of neurologic recovery.[53] Progressive neurologic deficit caused by a bullet in the spinal canal, a bony fragment, or a hematoma is indication for immediate surgery. The spine is stable in most of the cases, and stabilization rarely is required. Stauffer et al.[16] reviewed 185 cases of gunshot paralysis, half of which were treated with laminectomy and half with obser-

vation only. The authors recorded no appreciable return of neurologic function after surgery or nonoperative management for complete lesions. In another study, no neurologic improvement after lumbar laminectomy compared with nonsurgical treatment was noted, and the authors of that study reported a high rate of complications.[54] In a more recent study of 246 patients, it was concluded that decompressive laminectomy plays no role in outcome for patients with gunshot wounds to the spine. Decompressive laminectomy was associated with higher rates of CSF leak and meningitis.[3] Waters and Adkins[55] reported statistically significant motor improvement after surgical decompression from the T12 to L4 levels compared with nonoperatively treated spines. However, they considered only patients with bullets lodged within the spinal canal. At more distal sites in the thoracic and lumbar regions, surgical removal of the bullet and decompression of the neural elements had no significant effect. Neither group experienced infections.

Not many studies have been published regarding the timing of surgery. Yashon et al.[56] operated on 80% of their patients during the first 24 hours after trauma, whereas Azevedo-Filho et al.[3] operated on only 16.9% during that timeframe. Significantly higher rates of infection were noted if decompression and laminectomy of the lumbar spine were
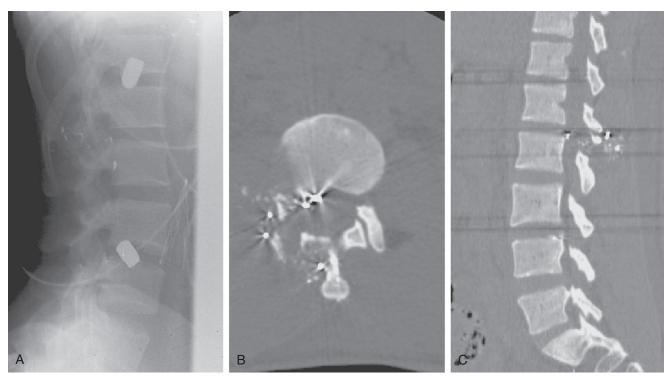

Fig. 58-4 *A,* Lateral view plain radiograph of a 25-year-old male patient shows two bullets at the level of L1-L2 and L4-L5. The patient was a victim of multiple gunshot wounds. *B,* Axial view CT scan reveals multiple fractures of posterior elements of L2 with bullet fragments in the spinal canal. *C,* Sagittal view CT scan shows fractures of the posterior elements of L1-L4. The patient's neurologic status was assessed as American Spinal Injury Association grade B. The patient was managed conservatively and achieved improvement to American Spinal Injury Association grade C.

performed 2 weeks after injury.[57] Irrespective of the level of injury, progression of a neurologic deficit and active CSF leak are indications for surgery. The patient should be medically fit to tolerate the surgery. For incomplete spinal cord injuries in the cervical and thoracic regions, mass effect to the spinal cord with structural lesion, which could be caused by bone, bullet fragment, or hematoma, are indications for surgery, whereas in the lumbar region, instability is the indication for surgery. The optimal time to perform surgery is 5 to 10 days after injury.[18]

In addition to the previously mentioned anatomic and biomechanical differences, infants and children have smaller blood volumes, differing anatomic proportions, less respiratory reserve, and the potential to injure growing structures.[58] These differences require a different approach for the proper management decisions to be tailored to the pediatric patient. The basic management of spinal injury remains the same. One study suggests that some children show improvement up to 2 years after the injury.[19]

High-energy gunshot wounds, which include rifle and shotgun wounds, cause a large zone of injury and massive tissue damage. Their treatment differs from the treatment of low-energy gunshot wounds.[18] Some authors have suggested aggressive débridement and decompressive laminectomy for such injuries; however, they also suggested that decompression is less likely to affect neurologic recovery.[22,59]

COMPLICATIONS

Pain is the most common and most problematic complication of spinal cord injuries caused by gunshot wounds.[60] Persistent pain can adversely affect the rehabilitation process. Conus medullaris and cauda equina level lesions have been linked to higher rates of pain.[61] Currently, little evidence exists that removal of the bullet is helpful in reducing pain.[60] Spontaneous discharges from the dorsal horn neurons cause pain and can be managed with orally administered neuroleptic medication, such as amitriptyline or gabapentin. Another option for unresponsive cases is a vascularized omental pedicle graft, which can be surgically transplanted to the affected cord level.[62] As a last resort, the dorsal root entry zone procedure, which surgically removes the nociceptive dorsal rootlets, has shown promising results.[61]

Acute and chronic infections are devastating consequences of gunshot wounds. Extraspinal abscess can form anywhere from the thoracic and peritoneal cavities to the retroperitoneal space and soft tissues. Spinal infections include meningitis, vertebral osteomyelitis, and perivertebral and epidural abscess. Fever should prompt lumbar puncture to rule out meningitis. If the infection is not subsiding with antibiotics, surgical débridement might be indicated (Fig. 58-5).

CSF fistula should be managed initially with subarachnoid drainage, and, if it persists, surgical exploration should
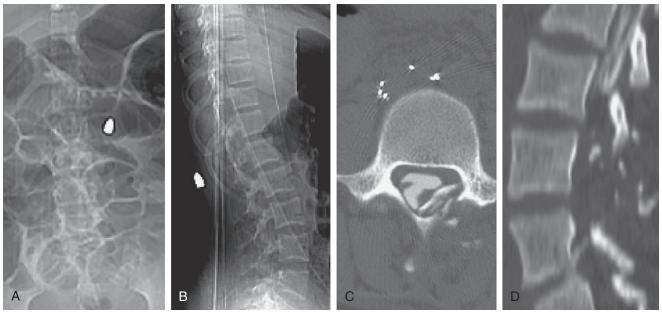

Fig. 58-5 Anteroposterior *(A)* and lateral *(B)* view plain radiographs of the thoracolumbar spine of a 17-year-old female patient who sustained a gunshot wound in her vagina. The bullet can be seen in the posterolateral aspect of the thoracolumbar region with fractured L4-S1 laminae. Axial *(C)* and sagittal *(D)* view CT scans reveal fractures along a trajectory extending from S1-L4 laminae, with a large bone fragment in the spinal canal at the L4 level. The thecal sac is compartmentalized below L4. The trajectory appears to have been through the spinal canal. The patient also had a colonic perforation, which later became infected, resulting in a pelvic abscess that required CT-guided drainage. This patient also had cauda equina syndrome.

be conducted. If a CSF fistula is detected more than 2 weeks after injury, surgery will be necessary because subarachnoid drainage is effective only if used during the acute stage.

Reports of bullet migration to the central nervous system have been cited by many authors as occurring as long as 11 years after initial injury.[62] Some have explained it on the basis of gravity or CSF flow.[45] Bullet migration is not always associated with neurologic complications, and if it is, retrieval should be assessed on an individual basis.

Lead poisoning usually is cumulative in nature, and reported incidences range from 2 days to 40 years.[63] Lead poisoning is, as such, a rare complication. Bullets in close proximity to facet joints and intervertebral disk spaces might be more likely to cause lead poisoning. Manifestations of lead poisoning include encephalopathy, peripheral neuritis, and colic. Encephalopathy might manifest as hypersomnolence, delirium, convulsions, and even coma. Signs and symptoms of lead poisoning include pallor, basophilic stippling on peripheral blood smears, and a "lead line" along the gingival margins. Blood lead levels should be measured to detect any increase. Bone marrow biopsy can confirm hematopoietic alterations. Treatment with chelating agents, such as calcium disodium edentate, should be started immediately, and carefully planned bullet removal should be performed at a later date.

Other rare complications are isolated bowel, bladder, and sexual dysfunctions. These conditions indicate the rare involvement of the autonomic nervous system.[45]

REHABILITATION

A team approach is required to achieve optimal rehabilitation of the patient. The team might include a physiatrist; neurologic surgeon; urologist; orthopaedic surgeon; pediatric surgeon; physical, occupational, and recreation therapists; nurses; social workers; and psychologists, depending on the needs of the patient. The majority of injuries result in complete paraplegia. Patients with thoracolumbar injuries and patients with incomplete injuries have the greatest recovery of motor function.[64]

CONCLUSION

Gunshot wounds commonly result from low-velocity weapons in civilians, but a trend toward high-velocity gunshot wounds is increasing. The initial focus for the patient with a gunshot wound must address all life-threatening injuries. Maintenance of ABCs is the most important. Tetanus prophylaxis routinely is indicated for all gunshot injuries. Steroids have no proven role in the treatment of gunshot wounds and should be avoided.

Surgery also plays very little role in the treatment of low-velocity gunshot wounds. It is very unlikely to improve the neurologic status of the patient. Surgery for spinal cord injuries resulting from low-velocity gunshot wounds are reserved for patients with progressive neurologic deterioration, persistent CSF fistulae, and sometimes for incomplete spinal cord injuries. Surgery for spinal cord injury victims might also be indicated to relieve active neural compression from a bullet, an intervertebral disk, or a hematoma within the spinal canal. Spinal instability rarely results from a civilian gunshot wound.

Cauda equina injuries resulting from low-velocity gunshot wounds have better overall improvement when compared with spinal cord injuries. As such, spine surgery in these cases, which actually entail peripheral nerve injuries rather than spinal cord injuries, might have a favorable result on long-term neurologic outcome. The decision to perform surgery should be made on an individualized basis.

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Management of Spine Fractures in Patients with Diffuse Idiopathic Skeletal Hyperostosis and Ankylosing Spondylitis

INTRODUCTION

Ankylosis of the spine provides a distinct set of challenges for the spine surgeon and health care practitioner evaluating and treating traumatic spine injuries. Ankylosis of the spine is defined as stiffening and immobility of one or more motion segments as a result of disease, trauma, or surgery. The two conditions most commonly associated with spinal ankylosis are ankylosing spondylitis (AS) and diffuse idiopathic skeletal hyperostosis (DISH). Patients with spinal ankylosis from either AS or DISH typically have a chronic history of back and/or neck stiffness that may or may not be associated with chronic pain as well. In such patients the acute onset of pain related to traumatic spinal injury may be difficult to distinguish. Moreover, these patients are at relatively increased risk of fracture as a result of relatively minor trauma. Although both AS and DISH have similar radiographic appearances and common clinical features, they represent distinct pathologic processes.

ANKYLOSING SPONDYLITIS

AS is an inflammatory rheumatic disease affecting the axial skeleton. The disorder was originally described in the 1890s and is still occasionally referred to eponymously as Bechterew's disease and Marie-Strumpell's disease. Hallmarks of AS include early sacroiliitis and inflammatory changes of the ligamentous structures of the spine resulting in progressive ankylosis, however other joints can also be affected. Extra-articular manifestations of AS may include acute uveitis, and up to 60% of AS patients have asymptomatic inflammatory bowel disease.[1] Cardiovascular manifestations include aortic insufficiency, pericarditis, and electrocardiogram (ECG) conduction abnormalities.[2] Rarely, pulmonary involvement can lead to upper lobe fibrosis and *Aspergillus* infection.[3] The typical clinical course consists of prolonged periods of disease quiescence characterized by back stiffness punctuated by periodic painful inflammatory flares. Uncommonly, severe disease involvement results in progressive thoracolumbar kyphotic deformity and functional incapacity. One outcomes study identified hip involvement or the presence of three of the following factors are predictive of severe outcome (sensitivity 50%) and practically excludes mild disease (specificity 97.5%): erythrocyte sedimentation rate (ESR) 30 mm per first hour, nonsteroidal anti-inflammatory drug (NSAID) unresponsiveness, limitation of lumbar spine, sausage digits, oligoarthritis, or disease onset at 16 years or younger.[4] Hip arthritis was associated with a 23-fold increase in risk of severe disease. Other studies have subsequently supported the correlation between hip involvement and disease severity in the spine.[5]

Patients can present at any age, but disease onset typically begins at younger than 30 years. Most patients report chronic back pain and stiffness that is more severe following prolonged immobility such as at night or during the early morning. Pain and stiffness characteristically improve with continued physical activity and are responsive to nonsteroidal anti-inflammatory medication. Additional medical history elements include previous diagnosis of iritis or uveitis, intestinal infections or inflammatory bowel disease, and family history of AS.

Physical examination may reveal multifocal tenderness at sites of enthesitis (inflammation of tendinous insertions), including the cervical, thoracic, and lumbar spine; sacroiliac joints; and other pelvic tendinous insertion sites, ribs, and heels. Diffuse costovertebral involvement can result in significant limitation of chest wall expansion, which can be identified by the Schober test. This test is performed by marking the posterior midline 10 cm above and 5 cm below the posterior superior iliac spines with the patient in the

standing position. With the patient in full forward flexion, the marked distance should be greater than 20 cm. Measurement less than 20 cm indicates reduced thoracolumbar motion and suggests the possibility of AS. Thoracic involvement by AS can result in limited chest wall expansion that may be identified by measuring circumferential chest expansion from full expiration to full inspiration. A minimum of 1-inch expansion is considered normal.

On radiographic evaluation, the hallmark of AS is sacroiliac joint erosion. Classically, joint appearance undergoes an evolution from early pseudowidening as a result of subchondral erosion, to subchondral sclerosis, to late joint space narrowing and ankylosis. Unfortunately, these joints are often poorly visualized using standard plain films, and 7 to 10 years of disease progression may be required prior to the appearance of significant joint changes using this modality. Computed tomography (CT) and magnetic resonance imaging (MRI) provide far greater anatomic detail, but diagnostic criteria for sacroiliitis and AS using these advanced imaging techniques have not been established. Early changes in the lumbar spine can be observed on plain radiographs as well and include diffuse demineralization, "squaring" of the upper lumbar vertebrae, and early syndesmophyte formation with discontinuous ligamentous calcification. The classic appearance of a "bamboo spine" with continuous paraspinal ligamentous calcification and mature syndesmophyte formation actually occurs in a minority of patients and is often associated with severe spinal involvement within the first 10 years of AS (Fig. 59-1).[6,7]

The pathogenesis of AS has been linked to genetic factors.[8] The clinical onset of AS is commonly observed in the second or third decade of life. Males are two to three times more likely to be affected than females.[9] The prevalence of AS among Caucasian populations has been estimated to be 0.1% to 0.2% and has been closely correlated with the population prevalence of the HLA-B27 major histocompatibility antigen on circulating lymphocytes.[10,11] Since the
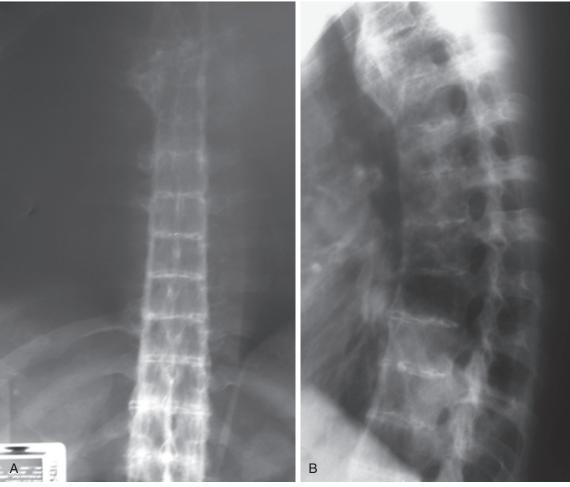

Fig. 59-1 AP *(A)* and lateral *(B)* radiographs of thoracic spine in patient with advanced AS but relatively preserved native thoracic kyphosis. Note the "bamboo spine" appearance on the AP radiograph and the continuous dense vertical midline crossing the thoracolumbar junction resulting from interspinous ligament ossification. Note the thin marginal syndesmophyte formation on the lateral radiograph in contrast to the thicker flowing calcification seen in DISH.

original discovery of a genetic linkage between AS and HLA-B27 was made in 1972, more than 100 separate disease associations with HLA-B27 have been made, including Reiter syndrome, psoriatic arthritis, inflammatory bowel disease, and reactive arthritis and uveitis. Among all known diseases associated with HLA-B27, the association with AS is one of the strongest. Eighty to 90% of patients with AS demonstrate the HLA-B27 haplotype, and the relative risk of an HLA-B27–positive individual for developing AS is 300 times that of the general population.[12] However, it is important to note that the presence of HLA-B27 is not diagnostic for AS. This haplotype is prevalent in the Caucasian population, and only 2% of patients with HLA-B27 develop AS.[12]

Elevated ESR and C-reactive protein (CRP) are present in approximately 50% to 70% of patients. However, disease severity has not been shown to correlate with elevated CRP and ESR. Mild elevation of serum IgA and high alkaline phosphatase levels may be present.[9]

Although the specific pathophysiologic mechanism for disease development has not been elucidated, enteric infections with *Klebsiella pneumoniae* and *Escherichia coli* have been implicated in those who are genetically susceptible.[13,14]

Treatment of AS consists mostly of physical therapy and nonsteroidal anti-inflammatory agents. Corticosteroids have a limited role, and chronic use is associated with osteoporosis, which can be a significant risk in the ankylosed spine. Radiotherapy is effective for treatment of refractory cases but has been associated with increased rates of acute myelogenous leukemia.[15]

DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS

DISH is clinically and radiographically similar to AS and often presents diagnostic confusion. DISH is considered to be in the category of disorders known as spinal enthesopathies, the others being Forestier disease involving the anterior longitudinal ligament in isolation, ossification of the posterior longitudinal ligament (OPLL), and ossification of the vertebral arch ligaments (OVAL), also known as ossification of the ligamentum flavum (OLF). These conditions may be different regional manifestations of a related pathologic process, that is, OPLL in the cervical spine, DISH in the thoracic spine, and OLF in the lumbar spine.[16,17] First described in 1950, DISH is a skeletal disorder characterized by ligamentous ossification of the anterolateral spine, most commonly the cervical and lower thoracic levels.[18] Affected patients may experience neck and back pain and stiffness with progressive loss of motion. Enthesopathy with inflammatory involvement of ligamentous and tendinous insertions in bone can be observed similar to AS and typically involves the elbows, knees, and heels. However, there is no involvement of the sacroiliac joints, and there is no association with HLA-B27. DISH is diagnosed and distinguished from AS and degenerative disk disease (DDD) based on several

radiographic criteria.[17] These criteria include: (1) presence of flowing calcification along the anterolateral aspects of at least four contiguous vertebral levels, (2) relative preservation of the intervertebral disk space and absence of degenerative disk changes, (3) absence of vertebral facet ankylosis, and (4) absence of erosion, sclerosis, or fusion of sacroiliac joints (Fig. 59-2). Similar appearance of marginal osteophytes may be present in patients with DDD, but these patients demonstrate radiographic and MRI evidence of disk degeneration such as collapse and cleft formation that are not typically present in DISH.

DISH is more commonly observed in individuals older than 50 years. Although studies have differed on the prevalence of the disease, it appears far more prevalent than AS with an estimated range of 6% to 12% of the North American population, and it is generally more common and more severe in men than in women (65% vs. 35%).[19] DISH appears more common in Caucasians

Fig. 59-2 Lumbar spine radiograph of DISH patient. Note the flowing calcifications along the anterior longitudinal ligament of four consecutive vertebrae and relative preservation of disk space. (Courtesy of Alexander R. Vaccaro, M.D.)

than in African Americans and Asians. The prevalence also appears to increase with age, with 28% of men older than 80 years demonstrating radiographic evidence of DISH.[20] Unlike AS, DISH has not yet been linked to a particular genetic antigen or marker. Nevertheless, a number of risk factors have been associated with DISH such as diabetes mellitus, obesity, hypertension, hyperlipidemia, and hyperuricemia.[21] A vascular derangement involving the nutrient arteries to the spine has also been implicated.[22]

Patients with DISH typically present with thoracolumbar back pain and stiffness. Similar to AS, stiffness is typically more severe in the morning and after periods of immobility. Cervical involvement can be associated with neck pain, stiffness, and dysphagia from prominent osteophyte formation. Peripheral enthesopathy can lead to a history of recurrent "tendonitis" affecting the shoulders, elbows, knees, and ankles. Patient complaints of intermittent sharp pain associated with bending and twisting motions are not uncommon.

The radiographic appearance of DISH is as described. For diagnosis, flowing syndesmophytes must be observed bridging four contiguous vertebral segments. Space exists between the syndesmophytes and the anterior border of the vertebral bodies. Syndesmophyte thickness varies from a few millimeters in early stages to 2 cm in advanced stages. Characteristically, the left side of the spine is significantly less involved or spared completely. This has been hypothesized as being secondary to the mechanical effect of the adjacent aortic pulsations. Additional radiographic manifestations of DISH in the axial skeleton can include ossification of the skull insertion of the posterior nuchal ligaments, the sacrotuberous ligament, and the symphysis pubis. Most common sites of ossification in the appendicular skeleton include the triceps insertion, quadriceps and patellar tendons, Achilles' tendon, and plantar aponeurosis. Advanced imaging studies are not typically required for general evaluation in the absence of trauma or neurologic deficit. Bone scans typically reveal diffuse nonspecific uptake of signal and are nondiagnostic.

Similar to AS, the mainstay of treatment for DISH involves regular physical exercise and periodic nonsteroidal antiinflammatory medication for painful flares of enthesitis.

SPINAL TRAUMA

Spinal fracture is a significant source of morbidity and mortality in patients with advanced forms of both AS and DISH. Vertebral fracture severity can range from occult, minimally displaced fractures without neurologic injury to fracture/dislocations with complete neurologic compromise. The issues with respect to evaluation and treatment of spinal trauma are similar in both AS and DISH. The presence of multiple contiguous fused segments results in a biomechanical lever arm that multiplies the stress of potential injury forces and results in significantly increased risk of fracture for any given level of injury. Dual energy x-ray absorptiometry (DEXA) measurements have revealed significantly reduced bone mineral

density in more than half of patients with AS. The increased prevalence of osteopenia and osteoporosis in this patient population further compounds the risk of fracture.[10,23] In a study of more than 1000 AS patients, the prevalence of vertebral fractures was 6.2% among male and 4.6% among female AS patients.[10] The prevalence of vertebral fracture increased to 14% among patients with disease duration for 42 or more years and was increased in patients with peripheral arthritis compared with those with axial involvement only. The incidence of occult vertebral fractures (without a clear episode of antecedent trauma) rose sharply in the third decade following disease onset.[10] Overall mortality rates of AS patients resulting from spinal trauma have been reported to be 0.7% to 2.8%.[24]

Despite being a widely acknowledged clinical problem, there are relatively few systematic studies of spinal trauma in either patient population. Most studies suggest that the lower cervical spine is the most common site of injury in AS with the second most common site being the thoracolumbar junction.[25–27] Although the risk of spinal fracture appears increased, there is no consensus regarding true rates or the magnitude of increased risk. It has been estimated that the incidence of thoracolumbar fracture in AS is four times greater than in the general population.[23] In AS, recognized patterns of thoracolumbar fracture include simple compression, transversely oriented shear fracture, and stress fracture associated with pseudarthrosis.[28] Among spinal injuries associated with AS and DISH, there appears to be a skewing of the distribution toward relatively more unstable three-column injuries and relatively increased rates of neurologic injury. The true rates of unstable injuries and spinal cord injury, however, again remain largely unknown. The rate of significant neurologic injury following cervical or thoracolumbar spine fracture in AS patients appears to be approximately 50%.[24,29,30] More severe three-column injury and spinal cord injury is typically associated with an extension-type mechanism (Fig. 59-3). Extension forces acting on the fused spinal segments lead to failure of the anterior column under tension and failure of the posterior elements under compression or shear. These three-column injuries are potentially very unstable, and any significant translation carries a high risk of causing or exacerbating spinal cord injury.

Although the risk of spinal fracture is generally considered to be increased in patients with DISH as well, there are relatively few specific studies or reviews of this problem in the literature. The three largest series report 29 fractures: 21 cervical, 6 thoracic, and 2 lumbar.[31–33] A review of all published cases suggests that more than half of the fractures in patients with DISH are transdiskal in location, roughly a quarter occur through the vertebral body, and a tenth involve both disk space and vertebral body. As in AS, most significant spinal fractures in this population are also the result of hyperextension injuries.[31–34] The risk of spinal cord injury also appears increased in spinal fractures complicating DISH. Although series are limited, reported incidences of initial or
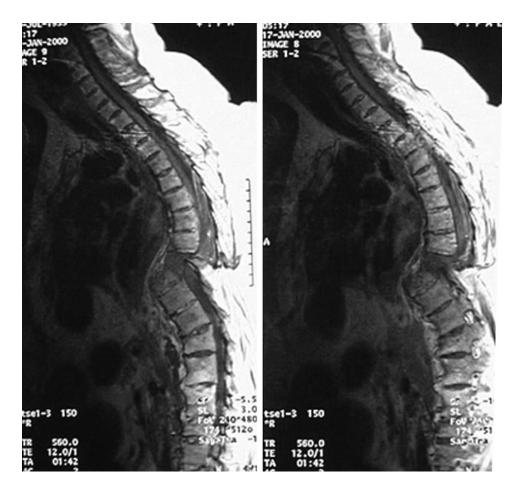
Fig. 59-3 Extension injury in patient with AS involvement of the thoracic spine, resulting in T8-T9 level shear fracture-dislocation and complete spinal cord injury.

delayed neural compromise range as high as 60% to 88%.[33,35]

DIAGNOSIS AND EVALUATION OF SPINAL FRACTURE

There are several factors unique to AS and DISH that make diagnosis of vertebral injury particularly challenging. First, limited patient mobility, coexisting osteoporosis, and hyperostoses of the spine may prevent adequate visualization of the spine using plain radiographs.[25,36,37] This can be a particular problem with fractures occurring near the cervicothoracic junction. Second, many fractures in these populations occur as the result of relatively minor injury (i.e., bed transfer or ground-level falls). Failure or delay in diagnosis may occur in as many of 33% of spine fractures associated with AS.[25] Clearly, a high level of suspicion is required in the evaluation of all AS and DISH patients with back pain complaints or even a history of mild trauma.

Clinical history should include specific questioning regarding any noticeable change in the patient's spinal alignment. Acute worsening of kyphotic deformity or change in sagittal or coronal plane alignment may have been recognized by the patient, friends, or relatives and suggests the presence of an underlying spinal fracture. Patients with advanced AS may report a sudden change in their field of view. A thorough evaluation of the patient's spine is indicated following significant trauma and should include visual inspection for soft-tissue swelling, ecchymoses, or other signs of local injury. Palpation should be performed for crepitus or spinal stepoff. Prior to any active manipulation or mobilization of the patient, a thorough neurologic evaluation should be performed. The presence of a new neurologic deficit strongly suggests the presence of an underlying spinal fracture until proven otherwise. Radiographic imaging of the spine is mandated prior to more active patient mobilization.

Patients with AS and DISH are susceptible to multiple noncontiguous spinal injuries.[38,39] Most authors recommend evaluation of vertebral fractures in these patients with CT imaging, including reconstructed images to evaluate both detailed fracture anatomy and overall spinal alignment.[25,36,37,40] Sagittal and coronal image reconstruction can be particularly helpful in identifying subtle displacements indicating the presence of an underlying fracture. MRI should also be considered to rule out an epidural hematoma compressing the spinal cord and to evaluate the integrity of the anterior and posterior longitudinal ligaments as well as the posterior spinal ligamentous complex.[32,35,41] MRI is also

useful in identifying nondisplaced fractures and development of posttraumatic pseudarthrosis.[35,41] Focal or linear appearance of increased signal on T2-weighted images suggests the presence of an acute fracture.

Hyperextension mechanisms have been associated with more unstable spinal injuries in patients with AS and DISH with potentially greater risk of spinal cord injury.[42,43] Evaluation of MRI images should therefore include careful inspection of the integrity of the anterior longitudinal ligament. Disruption of this ligament has been identified in up to 87% of cases and suggests a potentially more unstable injury.[41]

PATIENT MANAGEMENT

Special care must be taken with positioning and transferring patients with AS and DISH who have suspected spinal fracture. It cannot be assumed that neutral sagittal alignment represents the patient's native spinal alignment, and immobilizing a patient in this position may cause or worsen an existing spinal cord injury. Both the patient and any available individuals familiar with the patient should be involved in helping determine native alignment. Neurologic assessment should be repeated following any significant change in patient positioning. In patients with a native alignment of severe kyphosis, neurologic injury may be exacerbated through standard supine positioning of the patient, and consideration should be given to bolstering the patient in a lateral decubitus position. Applying standard spine injury protocols, including rigid cervical collars and supine positioning on a backboard, has been reported to contribute to further fracture displacement and has worsened neurologic deficits in multiple cases.[44,45] When performing closed reduction to improve alignment prior to surgical stabilization, bivector traction is recommended with careful radiographic monitoring to ensure that excessive distraction at the fracture site or aggravation of the deformity is not occurring (Fig. 59-4).

Frequent neurologic reassessment is critical in these patients, particularly during the initial 24 hours following an identified fracture. Progressive neurologic deficit suggests the possibility of fracture displacement or epidural hematoma and mandates reevaluation of patient positioning and emergent advanced imaging of the relevant region of the patient's spinal canal. MRI is the preferred study; however, CT myelography is an acceptable alternative in patients with contraindications for MRI.

In patients with stable, nondisplaced fractures without neurologic deficit, nonoperative management is an option and may include a cervical, cervicothoracic, thoracolumbar, or thoracolumbosacral orthosis, depending on the level of injury.[26,38,46] Prolonged bedrest with traction has been used in the past, but more contemporary patterns of treatment suggest that this option should be reserved for patients who unable to tolerate orthotic immobilization and medically unsuitable for surgical intervention. In patients with cervical level injury and significant native kyphotic deformity, a halo-vest orthosis may provide superior stabilization. In many cases, custom-molded orthoses are required to preserve the patient's native sagittal alignment. It is not advisable to attempt any correction to the patient's spinal deformity while bracing a healing fracture. Patients must be reassessed following application of any external orthosis to identify possible neurologic deterioration. Periodic reassessment is then required. Treatment with conservative methods such as traction or halo devices can lead to neurologic deterioration, failed union, or recurrent dislocation.[24,47–50]

SURGICAL MANAGMENT

Unstable three-column fractures, particularly extension-type injuries and shear injuries, should be strongly considered for early surgical stabilization. In the absence of medical contraindications for surgery, early surgery allows for more rapid patient mobilization and may reduce the risk of pulmonary complications.[51,52] Progressive neurologic deficit suggests the possibility of ongoing spinal cord injury from fracture displacement or instability and indicates the need for emergent surgical intervention to achieve immediate fracture reduction and stabilization. If an MRI can be obtained in a timely fashion, this study should be considered to identify the location of a possible epidural hematoma requiring decompression.[30,31,49]

The goals of surgery should be decided on prior to bringing the patient to the operating room and typically consist of decompression of neural elements, stabilization of the spinal column, or both decompression and stabilization. As previously mentioned, simultaneous deformity correction usually should not be attempted. Instrumented stabilization is nearly always an object of surgical intervention in these cases. Decompression may also be performed in the setting of incomplete cord injury, particularly if progressive neurologic deficit is observed and there is recognized epidural hematoma or discrete cord compression from intracanal fracture fragments. In cases of complete cord injury or cases of incomplete injury without evidence of compressive intracanal fragments careful realignment and spinal stabilization is all that is required. Delayed decompression can be performed if an initial complete injury turns out to be incomplete and persistent cord compression is identified.

In patients with advanced AS, a patient's native sagittal plane deformity may make intraoperative positioning quite challenging. Use of a standard Jackson table may not be possible, and it may be necessary to use a lateral decubitus position. Prone positioning over a flexed operating table is not recommended because of the possibility of inferior vena caval obstruction. In cases with severe deformity requiring posterior cervical spinal stabilization, surgery may be performed with the patient in a seated position, although there is an increased risk of air embolism. If a seated position is used, we recommend preoperative placement of a halo-vest orthosis (Fig. 59-5). Once the patient has been satisfactorily
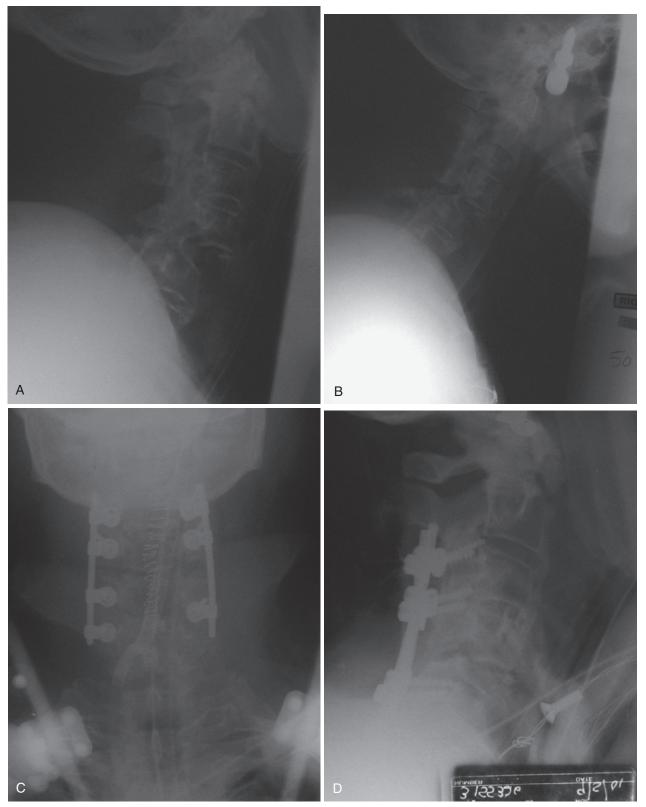
Fig. 59-4 *A,* Patient with DISH affecting the cervical spine following extension-type injury, resulting in C4-C5 fracture-dislocation and complete spinal cord injury. *B,* The injury was reduced with traction. *C, D,* A posterior stabilization procedure with lateral mass screw fixation was performed. (Courtesy of Alexander R. Vaccaro, M.D.)

Fig. 59-5 Preoperative positioning of AS patient with halo-vest orthosis in seated position prior to posterior cervical stabilization. (Courtesy of Todd J. Albert, M.D.)

positioned, the posterior half of the vest can be removed to allow access to the surgical site. Availability of multiple personnel to assist with positioning is essential, and generous use of carefully positioned bolsters or a surgical beanbag is recommended. During preoperative positioning the spine must be supported at all times as the weight of the body cephalad and caudad to the injury may increase deformity and contribute to injury of the spinal cord.[31]

CERVICAL SPINE STABILIZATION

Most commonly, unstable fractures of the cervical spine in patients with AS or DISH should be considered for combined anterior-posterior instrumented fusion (Fig. 59-6). If the fracture has occurred through a fused segment, the extended mechanical lever arms acting on the fracture site increase the risk of pseudarthrosis. In the setting of a cervical burst fracture without significant translation, anterior-only stabilization is an option with or without decompression-depending on the patient's neurologic status and the presence of ongoing spinal cord compression. Integrity of the posterior elements should

be assessed preoperatively on MRI, and care must be taken during anterior column reconstruction to avoid over-lengthening. Even with apparent anterior-column-only involvement, a long native fusion can lead to implant failure, and consideration should be given to supplemental halo-vest immobilization.

Both extension-type injuries and translational shear fractures are highly unstable, and combined anterior-posterior fusion is recommended. Posteriorly, the use of segmental instrumentation with multiple fixation points enhances immediate stability, and if there is a lengthy native-fused segment, little or no additional range of motion will be lost.[30,49]

THORACIC SPINE STABILIZATION

Similar considerations must be made in the setting of a thoracic-level injury. Most commonly, a posterior-only segmental instrumented fusion is performed for a three-column shear fracture-dislocation (Fig. 59-7, *A* to *C*). Given the strength of modern segmental instrumentation systems, even extension-type injuries can typically be stabilized through a posterior-only approach. However, care must be taken if posterior-only stabilization is to be performed in these cases.[30,31] Intraoperative distraction may result in excessive lengthening of the spinal column with spinal cord traction and worsening neurologic injury. Conversely, placing posterior instrumentation in excessive compression may create an exaggerated lordosis and exacerbate fracture deformity. For thoracolumbar injuries, the use of segmental instrumentation is strongly recommended. Both hook-and-screw systems allow for contouring around kyphotic deformity and balancing compression and distraction at multiple levels.[31,34] Anterior decompression of thoracolumbar-level injuries may be required in the setting of incomplete spinal cord injury and ongoing anterior cord compression, typically in the setting of a burst-type fracture pattern. If significant osteoporosis is suspected, the use of metal interbody cage devices is relatively contraindicated, and anterior-column reconstruction should be performed with structural allograft or autologous bone graft. Because of the increased risk of implant failure in these patients, strong consideration should be given to supplemental posterior-instrumented fusion as well.[30]

The ankylosed spine presents technical challenges to surgical stabilization. The landmarks for pedicle screw placement may be obscured. Intraoperative fluoroscopic guidance or surgical navigation may be of benefit in such instances. Alternatively, hook constructs are an acceptable alternative.[30]

The ideal construct length for fracture stabilization in AS and DISH patients has not been systematically investigated, and relatively few instructive studies are available from the literature. Success has been reported using fusion constructs from one to four levels above and below the injury level.[25,29,31] Factors influencing length of the fusion construct include
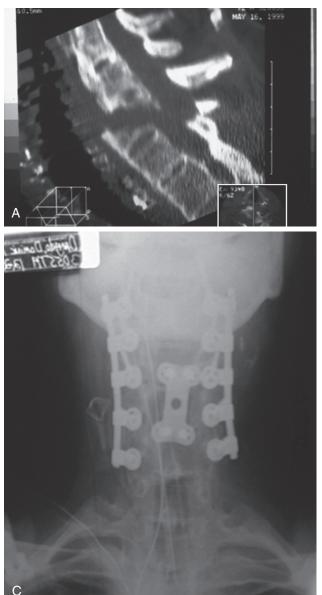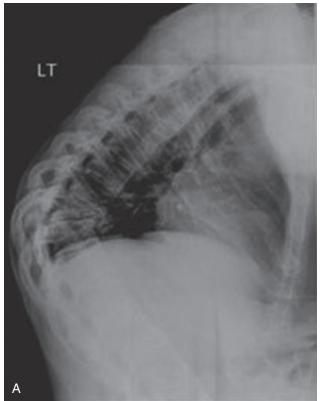
Fig. 59-6 *A,* Cervical extension injury in patient with AS resulting in unstable three-column fracture-dislocation and complete spinal cord injury. *B-C,* AP and lateral radiographs following anteroposterior stabilization of three-column injury in patient with AS involvement of the cervical spine. (Courtesy of Alexander R. Vaccaro, M.D.)
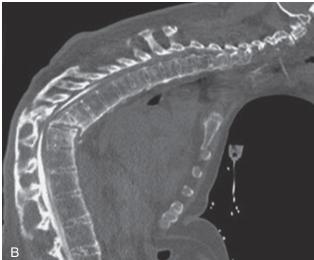
type of fixation device used (i.e., pedicle screws or hooks), severity of deformity, severity of injury, and underlying bone quality.

Although many investigators report the use of orthoses for postoperative immobilization, there is no consensus regarding the need for such immobilization, the type of orthosis, or length of immobilization required. Many published series have employed postoperative halo-vest immobilization for varying lengths of time until radiographic and clinical signs of fusion.[29,31,49] However, postoperative immobilization in a Pederson, SOMI brace, or cervical collar have been described as well with satisfactory results.[24,30] For AS or DISH patients with thoracolumbar fractures, most authors advocate the use of molded thoracolumbar orthoses postoperatively until radiographic and clinical signs of fusion appear.[29,31]

CONCLUSION

The ankylosed spine of AS and DISH clearly demands special consideration from treating health care professionals. Evaluation of these patients must be conducted with a high level of suspicion for injury even in the setting of apparently innocuous trauma. The unique spinal characteristics of these patients include the potential for significant kyphotic deformity, long segments of fused vertebrae, and osteoporosis. These characteristics merit consideration during every stage of patient evaluation and management. Patient positioning must take into consideration underlying deformity, and great care must be taken during any patient transfer or significant repositioning to maintain the patient's unique sagittal alignment.
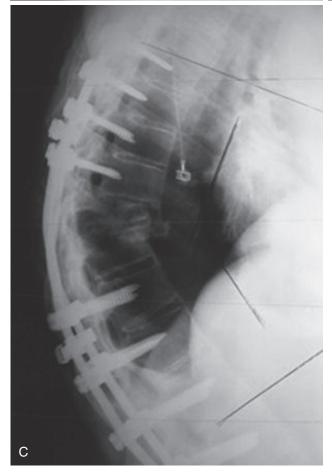
Fig. 59-7 T11-T12 fracture-dislocation in 60-year-old man with known AS who was injured in a motor vehicle accident and sustained a complete spinal cord injury. *A,* Preoperative lateral radiograph shows extensive ankylosis and native kyphotic deformity of the thoracolumbar spine and a sharp angulation at the level of injury. *B,* CT-myelogram sagittal reconstructed image showing three-column injury with compression failure of anterior and middle columns with distraction failure of posterior column and severe spinal cord compression. *C,* Postoperative lateral radiograph following open reduction and instrumented fusion. (Courtesy of Alexander R. Vaccaro, M.D.)

In addition to standard radiographs, advanced imaging with CT and MRI is essential for properly evaluating bone and soft-tissue injury in this population. Any significant deterioration in neurologic status merits consideration of repeat imaging of the spinal canal.

There is relatively sparse literature and no consensus regarding treatment. For stable injuries without neurologic deficit, conservative management using external orthoses is a reasonable option. For highly unstable extension-type injuries or shear-type fracture-dislocations, early surgical stabilization with instrumentation is preferred to allow rapid patient mobilization and to minimize the risk of medical complications. In all cases, the potential advantages of immediate stabilization must be weighed against the unique challenges of surgical management in this patient population, the health status of the patient, and the skill and experience of the operating surgeon.

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Surgical Treatment of Patients with Osteoporotic Fractures of the Spine

INTRODUCTION

Osteoporosis has become an area of rising health awareness because of the expanding aged population. The United States Bureau of Census estimates that 12% of the U.S. population older than 65 years has osteoporosis, a figure that translates to more than 36 million people. The rate is expected to grow and will likely include 68 million people by 2050. As osteoporosis becomes more prevalent, so will the number of patients being treated for traumatic injuries of the spine who also have osteoporosis.

Osteoporosis is associated with biologic and biomechanical alterations that have important consequences for the management of spine trauma. A preferential loss of cancellous bone occurs over cortical bone.[1,2] As a result, the ability of the spinal column to withstand axial loading diminishes in the osteoporotic spine. This has profound effects on anterior column implants. It can also significantly impact the biomechanics of bone fixation devices, such as pedicle screws and hooks. The normal anchoring dynamics are changed because of the loss of bone. This in turn changes the normal biomechanical considerations when designing spinal constructs. The biologic substrate for fusion, namely the existing bone mass of the spinal column, is markedly altered in the osteoporotic spine. Bone fuses at a slower rate and less robustly. To further complicate matters, the sites of normal autologous bone harvest often are degraded and therefore yield a limited volume of poor-quality tissue. In this chapter, we explore how the multitude of complex factors associated with osteoporosis interplay with the surgical management of spinal fractures, with particular emphasis on construct design and prevention of complications.

IMPLANT CONSIDERATIONS

The preferential loss of cancellous bone compared with cortical bone in the osteoporotic spine means that the cortical bone surfaces have much stronger implant purchase relative to the cancellous bone than in the normal spine.[1–4] As a result, the increased pullout strength that typically is afforded by pedicle screws compared with hooks decreases.[5–7] When bone is severely osteoporotic, hooks engaging the inferior lamina surface have a theoretical advantage over screws.[5,6,8] The inferior lamina has the most dense bone available in the osteoporotic spine.[8] Although this is universal from a biologic standpoint, it might be more important in the thoracic spine than in the lumbar spine based on anatomic variations. Lumbar pedicle screws have a long bony corridor that can be engaged. Within this corridor, 75% of the screw insertional torque is achieved at the neurocentric junction where the pedicle joins the vertebral body.[8] As such, the strength of the screws is not largely dependent on the cancellous bone of the vertebral body. Thoracic screws, by contrast, have a much shorter cortical tube to engage. In addition, the small diameter of the thoracic pedicle means that many of the screws exit the thoracic pedicle laterally, losing the insertional torque of the neurocentric junction. These features make them theoretically much more dependent on the cancellous bone of the vertebral body for purchase, which is precisely the bone lost in osteoporosis. Regardless of the location of a pedicle screw, it is particularly important that the screw diameter is large enough to engage the cortical bone of the pedicle.[5,7,9–11] Undersizing pedicle screws in the osteoporotic patient will place those implants at a significant biomechanical disadvantage such that the exertion of force on the screws during correction maneuvers can easily result in implant pullout and pedicle fracture.

To augment pedicle screw purchase in the osteoporotic spine, one can consider supplementing the vertebral body with bone cement and placing the implant as the vertebroplasty/kyphoplasty material hardens.[12–15] This strategy can improve bone purchase and increase the force required for implant pullout. Unfortunately, vertebral augmentation is not performed without risk, the most devastating of which is extravasation of cement into the spinal canal, which can result in neural injury. Although augmentation of pedicle

screw fixation with bone cement is a reasonable option for the osteoporotic population, it has been our preference to instead modify the construct design to accommodate the altered mechanical demands of the patients.

To achieve optimal surgical results, the surgeon must take into consideration the altered state of bone found in the osteoporotic spine. In some situations, hooks offer a potential advantage over screws. As stated, this is especially true in the thoracic spine. Unfortunately, laminar hooks enter the spinal canal and result in a loss of the ligamentum flavum, one of the most important aspects of the posterior tension band that resists flexion. The loss, combined with the decrease in anterior column support, can place the patient at risk for fracture and proximal junctional kyphosis.[16,17] This is further exacerbated by the stress riser experienced at the top of a rigid instrumented segment.

The stress riser at the cephalad and caudal construct terminus will affect rod choice. Most surgeons prefer a very stiff construct in cases of trauma to hold bony orientation until fusion occurs. With osteoporosis, increased stiffness can translate to construct terminus fractures. As a result, surgeons might prefer to choose a smaller diameter and slightly more flexible rod for osteoporotic patients to minimize the terminus stress and risk of new fracture.

The loss of cortical bone in osteoporosis also makes the endplate more prone to subsidence of interbody grafts. The thickest portion of the endplate is the outer edge. This apophyseal ring is the area of most dense endplate cortical bone. Interbody grafts ideally should spread load over the greatest surface area and engage the apophyseal ring. In this manner, force is both distributed and seated at the area of greatest bony resistance. An implant also should be made of material that matches the native bone modulus of elasticity as much as possible. Implants made of bone such as patellar wedges and femoral rings are ideal but are limited in supply and require on-site machining. Other useful devices are commercial materials with a modulus similar to bone, such as poly-ether-ether-ketone (PEEK) or carbon fiber. Metal interbody implants are more prone to subsidence and when used would ideally include an endcap to distribute load and fully engage the apophyseal ring.

The need for endplate outer cortex engagement to achieve optimal load distribution means that anteriorly placed interbody grafts are less prone to structural failure than are grafts placed by a posterior approach, such as with a posterior lumbar interbody fusion or transforaminal lumbar interbody fusion technique. With posterior approaches, which limit the size of the implant and its endplate coverage, the risk of graft settling and local loss of correction is more significant. This mechanical advantage must be weighed against the morbidity of a circumferential approach in an older patient.

Vertebroplasty and kyphoplasty can also play a role in both construct management and the primary management of trauma. In an older patient who has a traumatic vertebral fracture without significant canal compromise and intact ligamentous structures, it is recognized that vertebral augmentation can be an effective primary treatment. In a patient who has ligamentous disruption but does not have clinically relevant canal compromise, vertebral augmentation can be an effective supplement to segmental posterior instrumentation and fusion. As such, it might avoid the need for and morbidity of an anterior approach and interbody graft placement. In cases of canal compromise, vertebral augmentation has a limited role, if any.

Vertebral augmentation is a consideration in badly osteoporotic levels that are at risk of collapse above a construct because of the stress riser at the top of the construct. This is a very reasonable consideration despite the added risk of vertebral augmentation. Until more data are available, it is difficult to recommend this as a routine practice.

BONE GRAFTING AND FUSION

Fusion can be a challenge in cases of osteoporosis plus traumatic fracture. Normal bone graft harvest sites often contain a limited quantity of poor-quality bone because of the fatty replacement of cancellous bone observed in association with osteoporosis. This combined with a biologically inferior fusion bed means that the surgeon is uniquely challenged to provide the patient with the long-term success of solid bony ankylosis. The use of local bone from any osseous decompression procedure is critical and can be combined with other autogenous bone sources. Ribs can be a useful source when hip is not available or adequate. Use of the ribs is especially attractive for autograft harvest when a thoracolumbar approach is part of the reconstruction. In rare cases, autogenous fibula can be harvested. Because of the morbidity of autogenous harvest in an older population, osteobiologic agents have taken on an ever important and growing role. Bone growth modulators, including bone morphogenic proteins, demineralized bone matrix, and other agents that harbor some form of osteoinductive capability, have become increasingly available and are particularly useful for this group.[18] Internal or external electrical bone stimulation also is used to assist with osteoinduction. Osteoconductive products continue to play a pivotal role in supplementing whatever primary fusion modality exists.

CONSTRUCT PLANNING

SURGICAL APPROACHES

The scope of trauma is too broad to cover in a single chapter because of the unique considerations for each type of injury. In this chapter, we focus on general construct considerations and construct terminus design. The surgeon is provided with a set of guidelines to address the full spectrum of trauma rather than an encyclopedic set of rules for each type of fracture.

The first consideration in construct planning is to assess the approach needed to correct the fundamental biomechanical

failures caused by the trauma while effectively decompressing neurologic elements. If anterior column insufficiency occurs or if clinically relevant spinal canal compromise is present in a patient with osteoporosis, the anterior column must be surgically addressed. If no posterior injury occurs or if the spine is internally well supported, such as in the thoracic spine, anterior reconstruction alone can be considered. If the patient is severely osteoporotic, real risk of graft and anterior construct subsidence exists. One consideration is to extend the anterior construct an additional level above and below the injured vertebral body with interbody grafts (Fig. 60-1). In doing this, load is distributed over additional segments thereby decreasing the stress on any single set of screws or implant. In many if not most cases, circumferential reconstruction is necessary despite the potential morbidity.[19,20] If a stand-alone anterior construct is chosen, it is critical that bicortical purchase is obtained because of the poor cancellous bone quality.[21]

In the cervical and lumbar spine, the anterior approach is the only real option for the anterior column. In the thoracic spine from T3 to L1, the lateral extracavitary approach is an attractive technique for anterior column decompression and interbody reconstruction performed through a single posterior approach. This approach is our strategy of choice for this population when the anterior column problem lies between the T2-T3 disk and the L1-L2 disk. In addition to avoiding the potential morbidity of an anterior approach, including pulmonary compromise, the lateral extracavitary technique allows segmental posterior instrumentation and fusion to be performed, yielding a three-column spinal reconstruction through a single incision (Fig. 60-2).

Posterior approaches can be used alone in the absence of clinically relevant canal compromise when no anterior column insufficiency is present and if no major sagittal plane imbalance must be considered. Posterior-only approaches are acceptable with vertebral body loss if significant reconstitution of vertebral body height is present with postural correction and if that height can be maintained by a simultaneous vertebral body augmentation procedure.
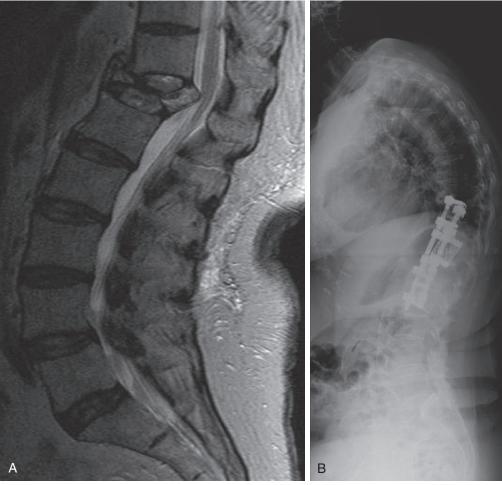
Fig. 60-1 *A,* Burst fracture of L1 requiring corpectomy in a patient with severe osteoporosis caused by chronic use of steroids. The patient also had a complex infected posterior wound that precluded a circumferential approach. *B,* To distribute load, the construct was extended from T11 to L3. A vertebral body replacement cage was placed at the corpectomy site (L1) and anterior diskectomy and interbody grafts were placed at T11-T12 and L2-L3. An anterior construct shared load over four rather than two vertebral bodies.
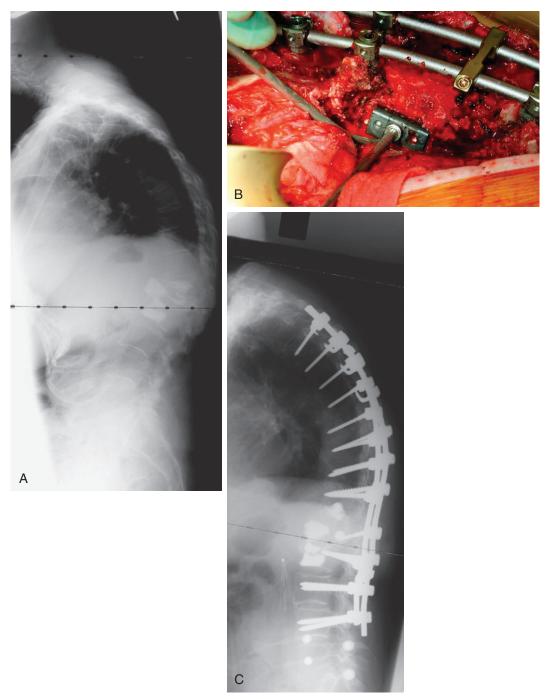
Fig. 60-2 *A,* Osteoporotic fractures of T12 and L1 for which vertebroplasty failed. The patient had progressive kyphosis and conus compressive symptoms. Poor lung compliance rendered an anterior approach difficult. *B,* Lateral extracavitary approach allowed circumferential access from an all-posterior approach. *C,* Posterior reconstruction could be accomplished at the same time, reestablishing sagittal balance.

As previously mentioned, many of osteoporotic patients being surgically treated for spine trauma require a circumferential approach to optimize load sharing and improve the chance for long-term solid ankylosis. This can be accomplished with an anterior–posterior procedure or the lateral extracavitary technique.

GENERAL CONSTRUCT PRINCIPLES IN OSTEOPOROSIS

It is counter intuitive that in older, sicker osteoporotic patients with traumatic or other spine reconstructive needs, more is better. In general, this is precisely the case.[22] In osteoporotic constructs, more implants will share load,

avoiding excessive stress at any one bone-metal interface. This ultimately makes the construct more stable and less prone to implant pullout or failure. In addition to maximizing the implant interfaces to distribute load, the liberal use of interbody grafts will significantly decrease the stress on posterior fixation devices.[4,23] Interbody graft placement approach and material choices should be considered based on the points brought up earlier. When possible, bicortical screw purchase should also be used to improve pullout strength. This is important with anterior constructs and screws in the sacrum. In both cases, the loss of cancellous quality renders unicortical purchase insufficient.[23,24]

CEPHALAD CONSTRUCT MANAGEMENT

Management of the cephalad end of the construct is important because the cephalad end is very prone to "topping off" or kyphosis of the segment above the fusion because of an osteoporotic fracture. Although this cannot be completely avoided, the risk can be minimized by considering the unique biomechanical forces on the spine and the general principles of construct planning previously described. One important issue is sagittal balance. Restoration of sagittal alignment is the single most important factor in predicting clinical success in adult spine reconstruction.[25] Establishing sagittal balance is important in the long term when performing spinal trauma reconstruction. In the short term, sagittal realignment will reduce stress on the construct terminus and improve the likelihood of successful arthrodesis. In general, we assure that osteoporotic constructs have the terminus away from spinal curve junctional zones and the apex of thoracic kyphosis. In general, we end the cephalad aspect of lumbar constructs at L2 or below. Constructs that terminate above L2 are at risk to top off at the thoracolumbar junction. In general, they should be taken to the lower structural thoracic spine if reasonable bone purchase and good sagittal alignment are present. If the bone quality is very poor or sagittal alignment is a concern, extending the construct into the upper thoracic spine should be considered if the patient is medically able to withstand a longer procedure. When extending the construct to the upper thoracic spine, we prefer to use a "blended construct" at the cephalad extent with screws on one side and hooks on the other. This strategy takes advantage of the good bone purchase of laminar hooks on the cortical bone of the inferior lamina and the tension band preservation of pedicle screws (Fig. 60-3).

In the cervical spine, it is important to end the construct in a position that is mechanically stable and has adequate bone purchase. This means that sagittal alignment must be within acceptable parameters, the tension band is preserved at the apex of instrumentation, and the cephalad bony terminus implants have adequate purchase. This can be a challenge in the cervical spine where lateral mass screws have limited purchase power. Here again, hooks can be considered on one side of the construct. Customized cervical hook systems are now available for such use. Cervical pedicle screws can be used, but the risk of spinal cord or vertebral artery injury has limited their role. When improved bony purchase is needed, the use of pars/pedicle screws in C2 can prove very useful.[26] In all cases, anterior interbody grafts can be valuable in unloading posterior implants and maintaining correction. This also has the additional advantage of placing graft in the ideal biologic environment to promote fusion.

CAUDAL CONSTRUCT MANAGEMENT

The caudal construct terminus in osteoporosis is primarily at risk for implant pullout and the resultant loss of sagittal plane alignment. Other concerns include the risk of caudal fracture at the lowest instrumented level. To minimize the risk of caudal end failure in trauma constructs, it is important that sagittal alignment is restored. This will minimize cantilever forces that exceed those normally generated on the caudal end of a construct.

In the lumbar spine, some have advocated supplementing a caudal screw with a hook at the same lamina to minimize screw strain and pullout.[27,28] Although this is a reasonable method to decrease caudal screw strain, it comes at the cost of tension band destruction at the caudal construct end. We prefer to preserve this to minimize the risk of late distal junctional kyphosis and to supplement the caudal end with an interbody graft when possible. This can be accomplished by using an anterior lumbar interbody fusion or transforaminal lumbar interbody fusion and/or posterior lumbar interbody fusion technique. We have also found a great deal of value in minimally invasive anterior interbody supplementation via the direct lateral trans-psoas approach. Through a very limited incision, multiple levels of interbody grafting can be performed from the caudal thoracic spine down to L4-L5 in some patients. The distal limiting factor for the use of this technique is obstruction by the ilium. This has the advantage of dramatically decreasing the load on the caudal screws while placing graft in an ideal environment to promote fusion.[23,29,30] This is critical because the caudal construct is at high risk for pseudarthrosis because of the cantilever strain experienced distally.

When the construct must extend to the sacrum, large cantilever forces place the construct at great risk for implant pullout, loss of sagittal alignment, and pseudarthrosis. We have also recently treated patients who developed severe fixed sacral kyphosis after lumbosacral fusion because of sacral insufficiency fracture. Such a fracture presumably developed from the large cantilever loads imposed on the sacrum in a patient with a relatively weak bony sacrum. To minimize the risks of distal construct failure, sound mechanical principles are essential. Sacral screws must be bicortical. They should also be offset with an interbody graft if possible to both unload screws and improve fusion.[23,24,29,31] Failure to adhere to these principles can lead to sacral implant pullout, lumbosacral pseudarthrosis, and loss of sagittal correction (Fig. 60-4).
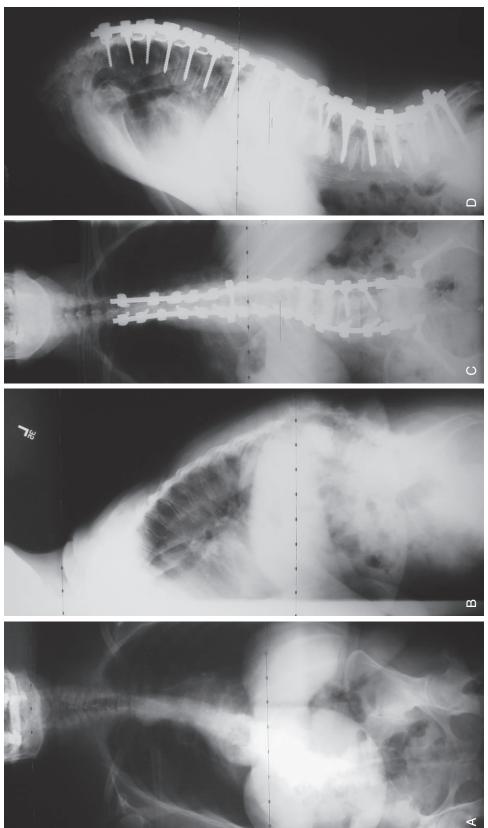
Fig. 60-3 Osteoporotic fractures and kyphoscoliosis with severe trunk shift (*A* and *B*) were corrected with a lateral extracavitary resection and posterior fusion (*C* and *D*). Note the hooks on one side of the construct and screws on the other, creating a blended construct that minimizes cephalad construct complication risks.
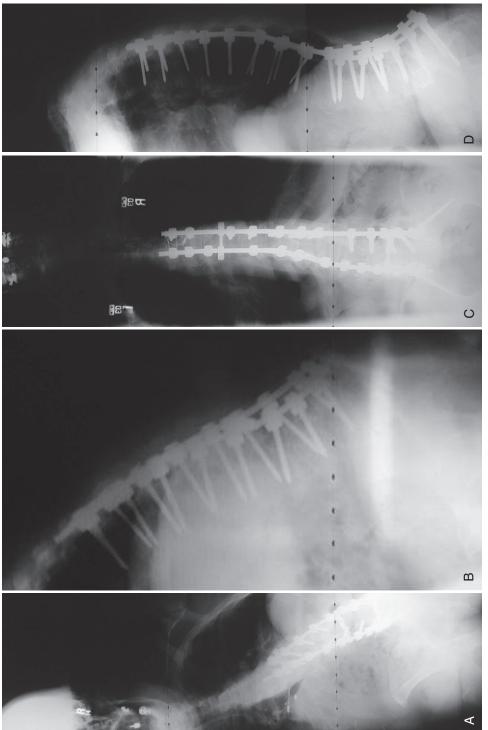
Fig. 60-4 *A* and *B*, Traumatic fracture repair to the sacrum without L5-S1 interbody graft or iliac fixation. This results in caudal S1 pullout and pseudarthrosis with severe coronal and sagittal decompensation. This was repaired with a pedicle subtraction, L5-S1 transforaminal lumbar interbody fusion and iliac fixation.

A construct that ends in the osteoporotic sacrum exposes this structure to large cantilever forces. S1 pedicle screws also create a "blade plate" type force in the sacrum. This combination is prone to insufficiency fracture.[32] As stated, this can result in pain and, at times, sacral kyphosis (Fig. 60-5). To avoid this, we recommend iliac fixation in any construct, trauma or other, that involves five or more vertebral levels.[29,31]

CONCLUSION

Patients with osteoporosis experience changes in bone quality and bone architecture that result in altered biomechanics and healing during traumatic spinal reconstruc-

tion. It is important that the spine surgeon appreciate the differences between this group of patients and those with traumatic injuries who do not have osteoporosis. The surgical plan must account for the differences in fixation strength, anterior interbody support, and bone healing capability. By applying common principles and techniques of spinal reconstruction in new combinations, the biomechanical disadvantages of surgery in the osteoporotic spine can be overcome in most cases.

Continued

Fig. 60-5 *A* and *B,* Traumatic pars fracture and spondylolisthesis that had instrumentation to the sacrum. An insufficiency fracture resulted in a sacral kyphosis with loss of sagittal balance.
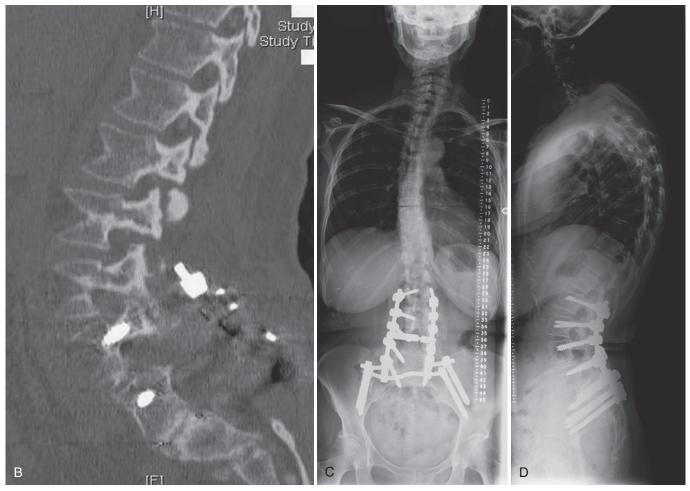
Fig. 60-5, *C* and *D,* This was repaired with a sacral-pelvic dissociation osteotomy and fusion.

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Surgical Management of Post-Traumatic Spinal Deformity

INTRODUCTION

Each year, approximately 10,000 Americans will present with a spinal cord injury and approximately 150,000 to 160,000 will present with a spinal column fracture.[1–3] These injuries can be devastating to both the patient and the family—especially when a spinal cord injury is present—and result in a profound change in the quality of life. Consequences can be seen both in the short term and also in the long term as complications develop over time and lead to further deterioration in function. Post-traumatic spinal deformity is one such complication and poses one of the greatest challenges in spinal surgery.

EPIDEMIOLOGY

The annual incidence of acute spinal cord injuries for patients admitted to acute care hospitals in the United States has been estimated to range between 3.2 to 5.3 per 100,000 persons; in other words, between approximately 10,000 and 17,000 new cases of acute spinal cord injury each year.[1,3] An acute spinal cord injury is estimated to be present in approximately 2.6% of all major trauma victims; of these between 43% and 46% result in complete loss of sensory and motor function below the level of the injury.[4,5] Approximately 55% of these injuries occur in the cervical spine, 30% in the thoracic spine, and 15% in the lumbar spine. Of the patients with injuries to the cervical spine, approximately 40% present with a complete spinal cord injury, 40% with an incomplete spinal cord injury, and the remaining 20% present with either no cord injury or only a root lesion.[4,6] The prevalence of spinal column injury is bimodal with peaks occurring in people between 15 and 24 years of age and in people 50 years and older with an overall mean age of 33 years.[1,3] With advances in safety standards, improvements

in emergency medical services, appropriate immobilization, and improved trauma care, the overall incidence of spinal cord injury has decreased over the years. In addition to a decreasing incidence of spinal cord injury, these factors have also resulted in an overall reduction in mortality, as well as a reduction in the progression of incomplete spinal cord injuries to complete ones.[1,7] As more patients survive their injuries, a greater number of patients are now faced with the long-term consequences of their spinal column fractures. Post-traumatic spinal deformity is thus becoming more prevalent, and therefore, correct management of this problem is now more important than ever.

PATIENT PRESENTATION

Post-traumatic spinal deformity usually presents following major trauma with resultant spinal column fracture; however, it can also present following minor trauma in patients with osteoporosis, ankylosing spondylitis, osteogenesis imperfecta tarda, and other endocrine or genetic disorders in which the bone quality is diminished. Patients may present after having undergone treatment including casting, bracing, or surgery but can also present having undergone no treatment at all. Regardless of the etiology of the post-traumatic deformity or the methods by which the initial injury was treated, the most common late complaints include one or more of the following: deformity in the sagittal and/or coronal plane, increasing pain, or an increasing neurologic deficit.

POST-TRAUMATIC SPINAL DEFORMITY

Post-traumatic spinal deformity is a common potential complication after spine trauma and its proper management depends on the thorough understanding of the normal alignment of the spine in both the sagittal and coronal planes.[7]

The thoracic, thoracolumbar, and lumbar segments of the spine contribute to the overall sagittal alignment of the spine. Thoracic kyphosis is measured from the upper (cephalad) endplate of T2 to the lower (caudal) endplate of T12 and normally ranges from +20 to +50 degrees of kyphosis with a mean of +35 degrees in normal adults.[8] Middle and lower thoracic kyphosis is measured from the cephalad endplate of

T5 to the caudal endplate of T12 and normally ranges from 10 to 40 degrees of kyphosis. By convention, kyphosis is a positive angle measurement and lordosis is a negative angle measurement. Thoracolumbar sagittal alignment is measured from the cephalad endplate of T10 to the caudal endplate of L2 and should be in neutral to slightly lordotic alignment. Lumbar sagittal alignment is measured from the cephalad endplate of T12 to the endplate of S1, and again kyphosis is a positive angle measurement and lordosis is a negative angle measurement. Normal lumbar lordosis ranges from –40 to –80 degrees with a mean of approximately –60 degrees in normal individuals.[8] In addition to understanding the normal thoracic, thoracolumbar, and lumbar sagittal alignment, it is important to understand how these parameters contribute to global sagittal alignment. Normal sagittal alignment allows the head to be positioned over the pelvis; in other words, a plumb line dropped from the center of the C7 vertebral body should pass anterior to the thoracic spine, cross the spine at T12-L1, and then travel somewhat posterior and pass over the posterior-superior corner of S1. If the C7 plumb line is anterior to the posterior-superior corner of S1, the patient is said to have a positive sagittal balance (i.e., she

or he is pitched forward), and if the C7 plumb line is posterior the posterior-superior corner of S1, the patient is said to have a negative sagittal balance. As is the case in normal sagittal alignment, in normal coronal alignment the head should also be positioned over the pelvis so that a plumb line dropped from the middle of the C7 vertebral body should pass over the center of the sacrum (i.e., the C7 plumb line and the center sacral vertical line should be one and the same). Traumatic injury to the spine can cause disruption of any of these parameters but typically leads to increased thoracic, thoracolumbar, and/or lumbar kyphotic alignment, and potentially positive global sagittal balance. Examples can be seen in Figures 61-1, 61-2, and 61-3. Although one could assume that increased thoracic, thoracolumbar, and/or lumbar kyphotic post-traumatic alignment would automatically lead to a positive global sagittal balance, many patients with post-traumatic deformity have normal balance secondary to compensatory changes in alignment by levels above and/or below the deformity, which is most accurately calculated by measuring the angle between the superior and inferior endplates of the vertebral bodies cephalad and caudal to the injured level.[9] Although at times it may be tempting to
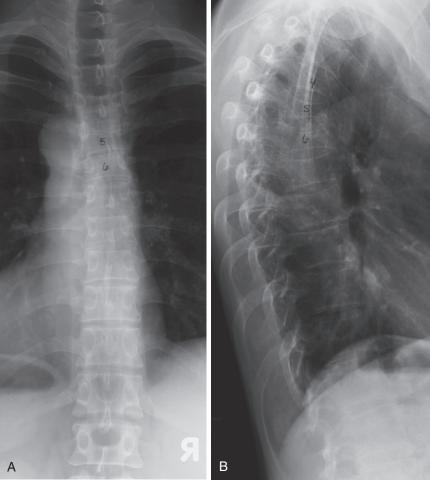

Fig. 61-1 Preoperative anteroposterior *(A)* and lateral *(B)* radiographs of a 43-year-old man who sustained multiple proximal thoracic compression fractures and presented with thoracic hyperkyphosis and pain.
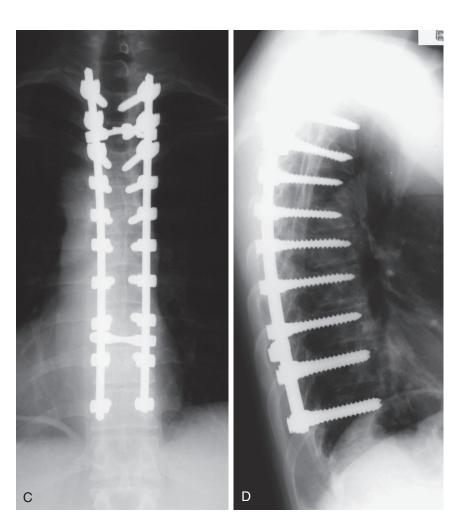
Continued

Fig. 61-1, He was treated with a posterior spinal fusion from T2 to T11 using thoracic pedicle screws as seen in the postoperative anteroposterior *(C)* and lateral *(D)* radiographs of the spine.

measure post-traumatic kyphotic deformity directly by measuring the fractured vertebra, this has been shown to be inaccurate.[7,9,10]

As mentioned previously, by far the most common post-traumatic spinal deformity is kyphosis, typically occurring in the thoracic, thoracolumbar, and occasionally lumbar spine and is most often due to the trauma itself.[2,7,9] In the thoracic spine, for example, a flexion-compression injury may result in a compression fracture of a thoracic vertebral body resulting in focal kyphosis at the level of the injury, an overall increase in the thoracic kyphosis, and possibly a positive sagittal balance. Although such fractures are unlikely to produce a progressive deformity because the posterior column is left intact, the local kyphosis may lead to abnormal spine biomechanics possibly accelerating the degenerative process.[9] If the injury is more severe—as in a severe burst fracture or a flexion-distraction injury, in which the anterior, middle, and posterior columns of the spine are disrupted—the post-traumatic deformity is likely to be greater and progressive in nature, especially if the injury occurs at the thoracolumbar junction or the lumbar spine where the spine is no longer supported by the ribcage.[7,9,11,12]

Although much less common, one must also remember that certain fractures—such as lateral compression or burst fractures—can lead to angular coronal post-traumatic deformity in addition to or instead of pure sagittal deformity.[7]

In addition to "acute" post-traumatic deformity resulting from the traumatic event itself, patients may present with "late" post-traumatic deformity resulting from other factors, some of which are related to the treatment of the injury (such as pseudarthrosis, implant failure, posterior-only surgery, short fusion segment, and prior laminectomy) and some that are unrelated to the treatment (such as osteonecrosis of the vertebral body, or Charcot spine). Patients with a pseudarthrosis may present with a progressive spinal deformity, pain localized to the surgical area, and radiographic evidence of nonunion and instrumentation failure/fatigue; it is paramount that in such situations an occult infection be ruled out because sometimes a deep infection can lead to a symptomatic pseudarthrosis. Implant failure, whether resulting from a pseudarthrosis or from other underlying etiologies including excessive forces at the implant/bone junction, surgeon error, patient noncompliance, or weak/osteoporotic bone, can also result in progressive post-traumatic deformity.

Implant failure can occur after both anterior and posterior instrumented fusions and often requires revision surgery. Aside from pseudarthrosis and implant failure, the other surgical factors that have been implicated in progressive post-traumatic kyphosis following the initial treatment of a spine fracture have been posterior-only surgery, a short fusion segment, and a prior laminectomy.[7,13–16] Progressive kyphosis of 10 degrees or more has been observed in approximately half of patients treated with short fusion segments[4] and can gradually worsen and eventually return to the preoperative position.[15,16] In at least one study, the authors advocate including five levels when performing a fusion at the thoracolumbar junction for a thoracolumbar fracture and avoiding a laminectomy at the area when possible.[15]

Osteonecrosis of the vertebral body (known as Kümmell's disease) can also lead to post-traumatic deformity. It is a rare spinal disorder in which osteonecrosis of the vertebral body develops in a delayed fashion after minor trauma to the spinal column.[17,18] As in osteonecrosis of the femoral head, in Kümmell's disease the vertebral body is prone to collapse once osteonecrosis has occurred and this in turn can lead to progressive deformity. Spinal cord injury resulting in paralysis and lack of sensation below the level of the injury may result in a rare complication of spinal cord injury known as Charcot spine. This disorder, also known as neuropathic spinal arthropathy, can lead to post-traumatic deformity and can be seen in patients treated both operatively and nonoperatively. The deformity is a consequence of the insensate nature of the vertebral elements below the level of the injury resulting in abnormal movement between the vertebrae, destruction of the joint surfaces, fracture of the subchondral bone, vertebral collapse, and ultimately a "ball-and-socket"
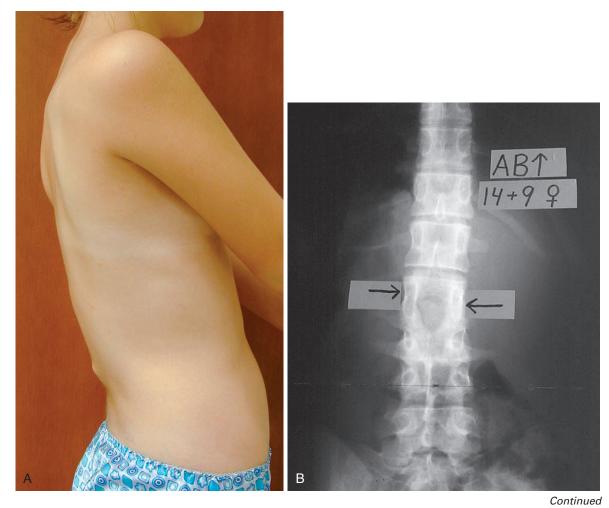
Fig. 61-2 Preoperative clinical photograph *(A)* and anteroposterior *(B)* and lateral *(C)* radiographs of the spine in a 14-year-old girl who was involved in a motor vehicle accident 1 year prior to presentation and sustained an L2 Chance fracture. Following her injury, she was treated with a TLSO for 6 months, and presented to our institution with significant segmental kyphosis with compensatory hyperlordosis below the level of injury *(B and C)*. She underwent a posterior spinal fusion from L1 to L3 with a L2 pedicle subtraction osteotomy with approximately 55 degrees of correction and restoration of normal sagittal contours as seen in the postoperative clinical photograph *(D)* and anteroposterior *(E)* and lateral *(F)* radiographs of the spine.
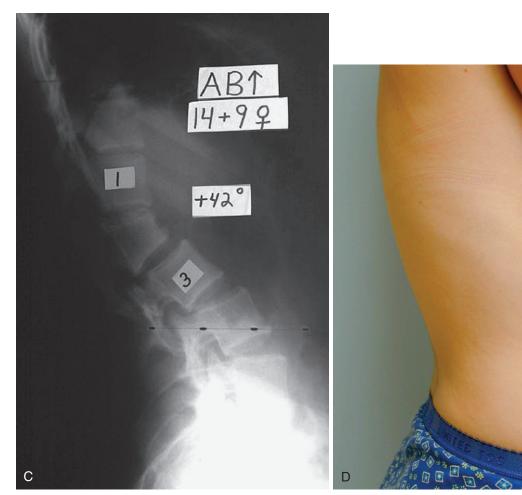
Fig. 61-2, cont'd *C* and *D*

pseudarthrosis. Charcot spine can progress rapidly and typically presents with progressive kyphosis, flexion instability, and loss of height.[7,19–24]

PAIN

Pain is one of the most common symptoms of post-traumatic deformity. The pain typically occurs at the level of the deformity and is typically constant and aching in character; the pain is thought to be caused by the abnormal spine biomechanics at the level of the spinal column deformity, resulting in altered forces being placed on the soft tissues and surrounding structures.[7,25] Studies have shown that patients with a focal kyphotic deformity equal or greater than 30° are an increased risk for chronic pain in the region of the increased kyphosis.[25–27] As the vertebral levels both above and below the deformity degenerate prematurely because of the altered spine biomechanics, patients may also complain of pain adjacent to the apical level of the deformity. Studies suggest that surgical correction of the deformity can at least in part improve pain symptoms related to the deformity. In two

studies by Bridwell et al.[28,29] on complications and outcomes of pedicle subtraction osteotomy for fixed sagittal imbalance, patients had a statistically significant improvement in pain visual analog scale following surgery, although in each of the two studies only a small subset of their patients had spinal deformity secondary to trauma. Similar results were seen in a study on the functional outcome and radiographic correction after spinal osteotomy by Ahn et al.,[30] but again only a subset of the patients in that study had post-traumatic spinal deformity. Kostuik and Matsusaki[31] have shown that anterior stabilization with instrumentation and decompression for late post-traumatic kyphosis can lead to significant pain reduction in the majority of patients. Similar results were seen in a study by Bohlman et al.[32] in which an improvement in chronic pain was seen after late anterior decompression of thoracolumbar fractures with canal compromise. Although surgery may improve pain resulting from post-traumatic deformity, the success of surgical intervention in providing pain relief is not completely predictable, and, therefore, pain alone should not be considered an absolute indication for surgical treatment of post-traumatic deformity.
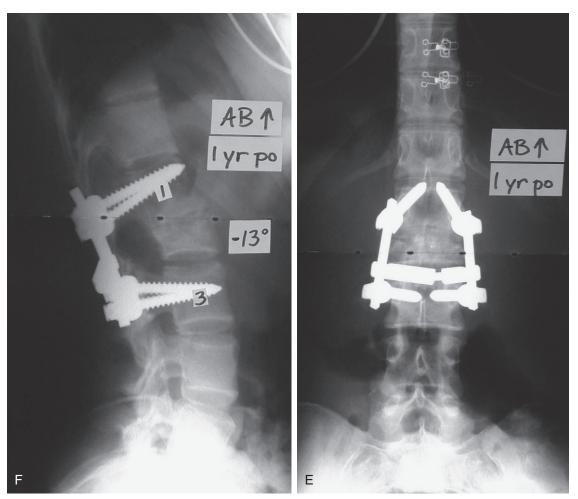
Fig. 61-2, cont'd *E* and *F*

NEUROLOGIC DEFICIT

Patients present with a new or increasing neurologic deficit in the setting of post-traumatic deformity typically for two reasons: development of post-traumatic syringomyelia and less commonly a neurologic deficit resulting from increasing deformity. Post-traumatic syringomyelia is common and its diagnosis has been increasing in frequency with the advent and wide availability of magnetic resonance imaging (MRI). Currently, post-traumatic syringomyelia accounts for approximately 25% of all cases of syringomyelia.[33] In the spinal cord injury population, 21% to 28% of patients can be expected to develop a syrinx anywhere from 1 to 30 years following the initial injury and 30% to 50% of patients will have evidence of spinal cystic change; however, only 1% to 9% of the spinal cord injury population will develop symptomatic syringomyelia.[33] The incidence of syringomyelia is similar following quadriplegia or paraplegia and no clear correlation has been found between the development of syringomyelia and the presence of spinal stenosis or complete versus partial spinal cord injury.[33] Post-traumatic syringomyelia may develop between 3 months and 34 years following spinal cord injury and usually presents with segmental pain and sensory loss followed by progressive asymmetrical weakness. The etiology is not well understood but is related to initial cord damage, persistent cord compression, arachnoiditis, altered subarachnoid space compliance, and perivascular flow.[33–35] Progression is typically gradual, although sudden deterioration (from hemorrhage into a syrinx) has been described.[33–35] Currently, post-traumatic syringomyelia is treated by a combination of cord untethering, arachnolysis, and duraplasty. Syringopleural or syringosubarachnoid shunting of the cyst in an alternative form of treatment, although shunting is associated with a failure rate of approximately 50%. No matter which of these procedures is used to treat syringomyelia, correction of the underlying deformity or cord compression should be performed first.[7,33–37]

Although less frequent than post-traumatic syringomyelia, post-traumatic deformity can also in it of itself be a cause of neurologic deterioration. Disruption of the vertebral column and supporting ligamentous structures may result in progressive kyphosis, stenosis, instability, and occasionally scoliosis, all of which can cause a new or worsening neurologic deficit

by direct compression or tenting of the neural elements without development of post-traumatic syringomyelia. In addition to direct compression, post-traumatic deformity can cause increasing mechanical stress on the spinal cord, which can in turn lead to arachnoiditis, cord tethering, and syringomyelia, all of which can obviously cause neurologic dysfunction. Both scenarios have been studied by Abel et al.[38] The authors reported on a group of patients who were found to have progressive loss of neurologic function without syringomyelia; in these patients, worsening neurologic deficits were thought to be due to progressive deformity, arachnoiditis, and cord tethering. In the same study the authors found that patients with less than 15 degrees of kyphosis and/or less than 25% post-traumatic stenosis were one half as likely to develop hydromelia than patients with residual deformity exceeding these values, which at least suggests that restoration and/or maintenance of "normal" spinal anatomy may prevent the development of post-traumatic syringomyelia and possibly secondary neurologic deterioration.[38]

SURGICAL CONSIDERATIONS

Surgical intervention for post-traumatic deformity is considered for a combination of the following indications: significant or increasing spinal deformity, increasing back and/or leg pain, "breakdown" at levels above or below the deformity, pseudarthrosis or malunion, and increasing neurologic deficit. A wide variety of procedures have been described to address correction of post-traumatic deformity including posterior-only procedures, anterior-only procedures, or combined
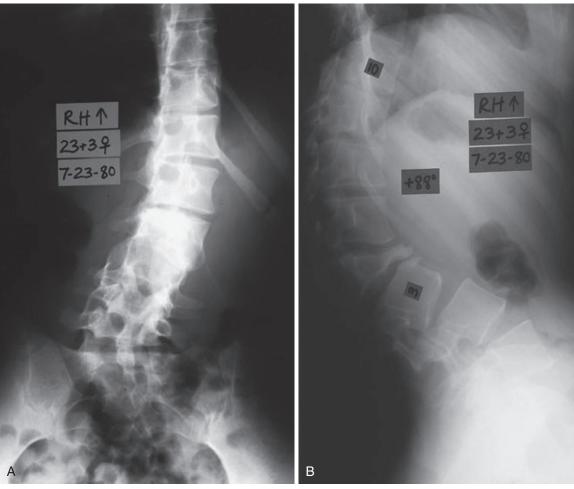
Fig. 61-3 Anteroposterior *(A)* and lateral *(B)* radiographs of a 23-year-old girl who sustained a three-column compression/flexion-distraction injury. She was treated at an outside institution with a combined anterior/posterior spinal fusion from L1 to L3. Postoperatively, she developed progressive spinal deformity and presented 18 years after her surgery with approximately 105 degrees lumbar kyphosis as seen in a clinical photograph *(C)* and anteroposterior *(D)* and lateral *(E)* radiographs of the spine. She was treated with a posterior spinal fusion from T10 to sacrum with L2 pedicle subtraction osteotomy and a staged anterior spinal fusion from T10 to sacrum with correction of her deformity and restoration of normal sagittal balance as seen in the postoperative clinical photograph *(F)* and anteroposterior *(G)* and lateral *(H)* radiographs of the spine.

anterior/posterior procedures. Regardless of the approach, the main goals of surgery are to decompress the spinal canal if neurogenic claudication or a neurologic deficit is present; to recreate normal sagittal contours and balance; and to maximize the chances of successful fusion by providing adequate support to the anterior column of the spine (to allow compressive forces across the anterior column) and sufficient instrumentation posteriorly (to provide tensile forces across the posterior column). This last factor is particularly important to remember because a posterior-only fusion in the presence of an existing kyphotic deformity—especially when combined with a positive global sagittal balance—will place significant tensile forces on the instrumentation and bone graft, and could ultimately lead to pseudarthrosis, implant failure, and/or progression of the deformity.

When surgery is indicated because of an increasing neurologic deficit or claudication, the involved neural structures have to be decompressed. Traditionally, this has been accomplished by performing a corpectomy through an anterior approach resulting in middle column decompression—which in addition to allowing a more direct method of decompressing the spinal cord than is possible through a posterior, pos-

terolateral, or transforaminal interbody approach—allows easier access to the anterior column, which can be difficult with the limited exposure provided by a posterolateral approach.[12,27,31,39] Several studies have also reported greater neurologic recovery after an anterior decompressive procedure in the setting of an incomplete deficit compared with posterolateral procedures even as late as two years after initial injury.[39-41] This finding, however, has not been corroborated by a recent review of the literature on the topic, which found that partial neurologic deficits had the potential to resolve irrespective of the approach used[42]; therefore, an anterior approach is not necessarily mandated when performing surgery for an increasing neurologic deficit as long as a middle-column decompression is performed and the spinal cord is adequately decompressed.

When performing surgery in the setting of post-traumatic deformity, it is of utmost importance not only to correct the focal deformity, but to consider the overall sagittal alignment and restore the normal sagittal contours and correct the global sagittal balance if necessary. The first step in deciding what factors need to be addressed during surgery is to assess the magnitude of the focal deformity, which can be due to
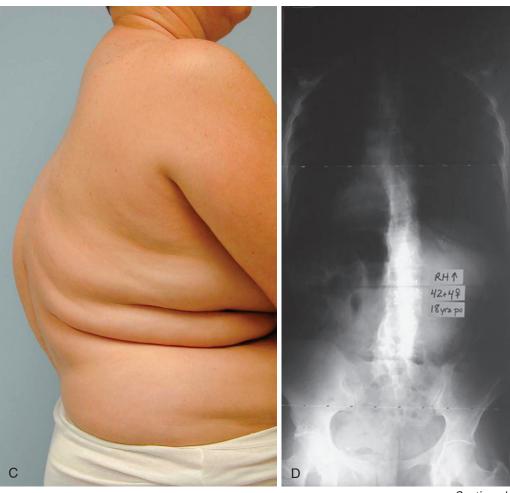

Fig. 61-3, cont'd *C* and *D*   *Continued*
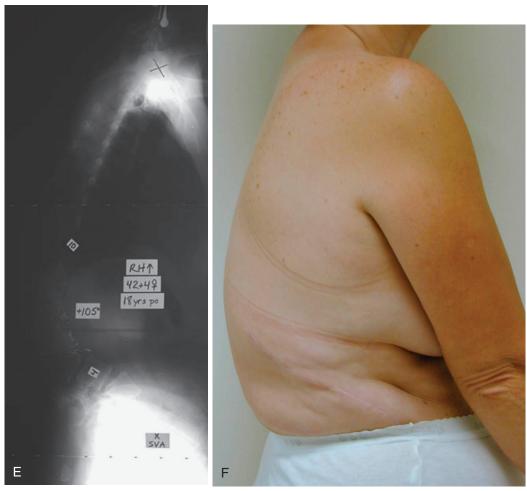
Fig. 61-3, cont'd *E* and *F*

fracture malunion following prior surgery, following nonoperative treatment, or following no treatment at all. Correction of focal deformity is important, not only because it can affect the overall sagittal balance, but also because studies have shown that patients with a focal kyphotic deformity equal or greater than 30 degrees are an increased risk for chronic pain in the region of the increased kyphosis.[25–27] Similarly, in at least one study, paraplegic patients with more than 15 degrees of kyphosis and/or more than 25% post-traumatic stenosis were more than twice as likely to develop hydromelia than patients without residual deformity exceeding these values.[38] In addition to assessing the magnitude of the deformity, the character of the focal deformity has to be evaluated because sharp angular kyphosis, for example, has to be treated differently than smooth kyphosis. The flexibility of the deformity also has to be assessed; this is typically based on a supine lateral hyperextension radiograph, but can also be based on a prone lateral radiograph. Once the nature of the local deformity is well understood, the next step is to determine the overall sagittal balance. Patients with a focal deformity, but with normal overall sagittal balance are said to have a type I deformity and will obviously have to be treated differently than those patients with a focal post-traumatic deformity and nonphysiologic or disrupted sagittal balance who are said to have a type II deformity.[43] Lastly, if prior surgery was performed and the deformity is the result of a malunion, the deformity has to be carefully screened for the presence of a pseudarthrosis; if one is present, a combined anterior/posterior revision procedure should be strongly considered to increase the chances of successful fusion following revision surgery.

Correction of a positive global sagittal balance depends on the flexibility of the deformity. A flexible post-traumatic deformity, which fully corrects on a supine lateral hyperextension and/or prone lateral radiograph, can typically be corrected by proper positioning on the operating table, followed by an instrumented fusion. An inflexible post-traumatic deformity, on the other hand, is much more difficult to correct and typically requires a combined anterior/posterior approach or a posterior-only approach combined with an osteotomy to correct the sagittal alignment. The two osteotomies that are used most commonly to correct sagittal deformity are the Smith-Petersen and pedicle subtraction osteotomies.
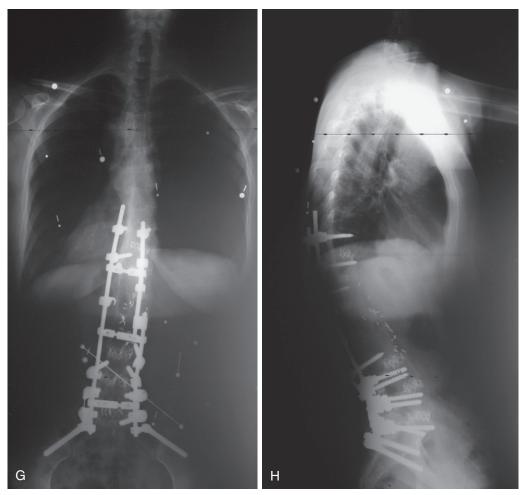
Fig. 61-3, cont'd *G* and *H*

The Smith-Petersen osteotomy was first described in 1945 for the treatment of kyphotic deformity[44] and has since then been modified by others.[45–50] It relies on shortening of the posterior column by resection of the posterior elements (ligamentum flavum and facets) at the desired level of correction with undercutting of the adjacent spinous processes. Sagittal correction is achieved through compression across the posterior column with concomitant elongation of the anterior column through the disk space and anterior longitudinal ligament (or osteoclasis through the vertebral body) with the axis of correction occurring through the posterior margin of the vertebral body. Because the osteotomy lengthens the anterior column, it may destabilize the spine if fusion does not occur and the instrumentation used to achieve compression across the posterior column fails; this potential drawback has led some to recommend anterior column structural grafting in conjunction with posterior Smith-Petersen osteotomies.[45,48–51] In addition, lengthening of the anterior column has been associated with superior mesenteric artery syndrome and even rare traction injury of one or more of the great vessels frequently resulting in death.[45,52–57]

A Smith-Petersen osteotomy results in approximately 1 degree of correction for each millimeter of posterior bone resected with an average correction of 10 to 15 degrees per level with overall correction reported in the literature ranging from 25 to 40 degrees.[43,45,49,52,53,56,59]

The pedicle subtraction osteotomy (also known as transpedicular cortical decancellation osteotomy) was first described by Thomasen,[60] although an earlier variant was described by Leong et al.,[61] and other authors have described a variation of the osteotomy referred to as the eggshell osteotomy.[62] The pedicle subtraction osteotomy is performed by first removing all posterior elements at the level of the correction—including the spinous process, lamina, superior and inferior adjacent facets, and pedicles—followed by decancellation of the posterior part of the vertebral body (and in the eggshell modification, removal of the posterior and lateral vertebral body walls) and hyperextension of the spine produced by a posteriorly based vertebral wedge fracture hinging on the anterior margin of the vertebral body. The osteotomy is closed by compression across the posterior column instrumentation or by extending the patient's position on the operating room table while taking care to ensure that neural elements are not compressed.[28,29,51,58]

Although the procedure is technically demanding and may result in substantial blood loss as a result of bleeding from the epidural venous plexus and cancellous bone, it provides several advantages. Unlike the Smith-Petersen osteotomy, in which the axis of correction occurs through the posterior margin of the vertebral body, the pedicle subtraction osteotomy results in segmental lordosis and correction of sagittal balance without elongation of the anterior column. Because the osteotomy results in bony apposition, the procedure provides greater stability and potential for union than a Smith-Petersen osteotomy. Additional advantages include the ability to obtain significant correction at a single vertebral level and to achieve both sagittal and coronal correction by resecting an asymmetric wedge of the vertebral body and cortex.[28,29,51,58] As a general rule, with a pedicle subtraction osteotomy, one can expect 30 to 35 degrees sagittal correction per level.[28–30,58,62–67]

Once the nature of the focal deformity—including the magnitude and flexibility of the focal deformity—and the overall sagittal balance are understood, a treatment plan can be developed. Type I post-traumatic deformity (i.e., focal deformity with maintenance of normal sagittal balance) and smooth post-traumatic kyphosis—whether in the thoracic or lumbar spine—can be treated with a single or multiple Smith-Petersen osteotomies depending on the magnitude and flexibility of the deformity. When the deformity is flexible, adequate correction may sometimes be obtained with proper positioning on the operating room table and osteotomies can be avoided as shown in Figure 61-1. Sharp angular post-traumatic kyphosis in the thoracic spine can typically also be treated with Smith-Petersen osteotomies; in the lumbar spine, on the other hand, a pedicle subtraction osteotomy is usually preferred as shown in Figure 61-2. Treatment of type II post-traumatic deformity (i.e., focal deformity with disruption of the global sagittal balance) is more complex and depends in large part on the amount of sagittal imbalance and nature of the focal deformity. Patients with minor sagittal imbalance (<2.5 to 5 cm) and smooth post-traumatic kyphosis, whether in the thoracic or lumbar spine, can be treated similar to patients with a type I deformity and smooth post-traumatic kyphosis and undergo a single or multiple Smith-Petersen osteotomies depending on the magnitude and flexibility of the deformity. Patients with major sagittal imbalance (>2.5 to 5 cm) and smooth post-traumatic kyphosis in the thoracic spine can typically be treated with Smith-Petersen osteotomies, although in our experience if a marked global imbalance exists, then three or more Smith-Petersen osteotomies usually are required. Patients with major sagittal imbalance and smooth post-traumatic kyphosis in the lumbar spine typically are treated with a pedicle subtraction osteotomy. Patients with minor sagittal imbalance and sharp angular post-traumatic kyphosis are usually treated with a Smith-Petersen or pedicle subtraction osteotomy when the deformity is present in the thoracic spine, and a pedicle subtraction osteotomy when the deformity is present in the lumbar

spine, as shown in Figure 61-3. Patients with major sagittal imbalance and sharp angular post-traumatic kyphosis typically require a pedicle subtraction osteotomy regardless of whether the osteotomy is present in the thoracic or lumbar spine.

When a long fusion construct is necessary to correct the post-traumatic deformity and it extends distally to the sacrum (or even the distal lumbar spine), anterior column support should be considered given the high pseudarthrosis rate observed when a thoracolumbar fusion is extended to the sacrum.[68,69] The use of anterior structural grafts in such situations not only aids in restoration and preservation of sagittal alignment but also creates load sharing between the anterior and posterior implants, increasing the chances of arthrodesis. The anterior column can be accessed directly through an anterior approach or indirectly through a posterolateral approach (more specifically through either a transforaminal interbody or posterior lumbar interbody approach), which may be more practical in the lower lumbar and/or sacral segments (i.e., L4, L5, and sacrum) because of the difficulty of reconstructing the anterior column in this region.[9] When combined with posterolateral fusion, both the transforaminal and posterior lumbar interbody approaches allow circumferential fusion, which is extremely useful, especially in patients in whom an anterior approach may be difficult or contraindicated.

OUTCOME

Results of treatment of post-traumatic deformity are generally encouraging and are dependent on the type of initial injury, duration of time between injury and deformity correction, and age and medical condition of the patient.[7] Keene et al.[13] found that patients with more than 40% vertebral body collapse at the time of surgery had poor results; in addition, patients operated on within 12 months of their initial injury appeared to have better outcome than those operated on a later date. Kostuik and Matsusaki[31] reported on 37 patients treated with anterior stabilization, instrumentation, and decompression for late post-traumatic kyphosis. Stable fusion was achieved in 36 of the 37 patients. The authors found that pain was reduced significantly in 78% of their patients; in addition, they found that patients with spinal stenosis who underwent decompression at the appropriate levels had improvement in their symptoms, and late neurologic improvement was noted in three of eight patients with paraplegia.[31] Similar results were noted by Malcolm et al.[25] who reported on 48 patients treated surgically for post-traumatic kyphosis 6 months or longer after the initial injury. Posterior-only procedures were done in 16 patients, anterior-only procedures were done in 12 patients, and combined anterior and posterior procedures were done in 20 patients. All patients treated with a

posterior or a combined procedure went on to a successful fusion, but only half of the patients treated with an anterior procedure went on to a successful fusion in contrast to the high fusion rate noted by Kostuik and Matsusaki.[25,31] Nevertheless, the authors found that pain was significantly reduced in 31% of patients and was completely resolved in 67% of patients.[25] In a study of 38 patients with late post-traumatic kyphosis treated with a single-stage posterior closing wedge osteotomy, Lehmer et al.[70] found that all patients went on to a successful fusion at latest follow-up and 93% maintained correction averaging 35 degrees. They found that 8 of 14 patients with a preoperative neurologic deficit improved postoperatively; in addition 76% of their patients stated that they would have the surgery again and 90% would recommend it to another.[70] Similar radiographic outcome was noted by Wu et al.[67] who found an average 38.8-degree correction of post-traumatic kyphosis following posterior decompression, transpedicular cortical decancellation osteotomy, and instrumentation and fusion of one segment above and below the level of a rigid post-traumatic kyphotic deformity. Favorable radiographic results following surgery for post-traumatic deformity have been noted in other studies.[71,72]

CONCLUSION

Trauma to the spinal cord and vertebral column is a devastating injury and can result in profound changes in the quality of life of both the patient and his or her family. The injury is often fraught with short- and long-term complications that may lead to further deterioration in the quality of life. Post-traumatic spinal deformity is one such complication and poses one of the greatest challenges in spinal surgery. The best treatment for post-traumatic spinal deformity is prevention with careful initial evaluation, close follow-up, and early intervention when needed. Once present, the treatment of post-traumatic deformity is challenging, and its success is dependent on careful patient selection and appropriate surgical intervention. Surgery should be considered in the presence of significant or increasing deformity, increasing back and/or leg pain, "breakdown" at levels above or below the deformity, pseudarthrosis or malunion, and increasing neurologic deficit. Regardless of the approach used to correct the post-traumatic deformity, the main goals of surgery should be to free the neural elements if neurogenic claudication or a neurologic deficit is present, to recreate normal sagittal contours and balance, and to maximize the chances of successful fusion by using appropriate bone-grafting techniques and adhering to basic biomechanical principles of providing adequate support to the injured columns of the spine, so that stability and correct sagittal and coronal balance can be restored. A thorough, systematic, and technically well-executed surgical intervention should hopefully allow successful outcome.

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Management of Post-Traumatic Syringomyelia

INTRODUCTION

d'Angers[1] first introduced the term syringomyelia in 1937 when he described a glial-lined cavity in the spinal cord that could communicate with the central canal or sub-arachnoid space.[2] Subsequently, cystic degeneration of the spinal cord in the setting of spine trauma became a well-recognized entity, as documented by numerous authors in the late 1800s.[3–5] Wagner and Stolper,[5] in 1898, provided a detailed pathologic description of a post-traumatic syrinx after postmortem analysis of the spinal cord of a patient with traumatic fractures of the fifth thoracic and first lumbar vertebrae.[6] They described extensive cavitation of the cord, both rostral and caudal to an area of contusion, which was in close proximity to an area of leptomeningeal thickening, lined by glial cells, and filled with a turbid gray fluid. More recently, Barnett and Jousse[7] highlighted the clinical syndrome of syringomyelia after spinal trauma; they reviewed a number of patients who experienced a delayed, ascending myelopathy months to years after initial injury.

Recent progress in the acute and chronic management of spinal cord injuries, immediate and long-term survival, and quality of life has accomplished dramatic improvements. This, along with advances in and increased availability of imaging techniques, has made post-traumatic syringomyelia a more prevalent and well-recognized cause of delayed deterioration in patients with spinal cord injuries (Fig. 62-1). Patients who might have improved or plateaued neurologically after spinal cord injury can present with progressive neurologic deterioration after months, or years, of clinical stability. The timely diagnosis and management of syringomyelia in this setting is critical to halt the neurologic deterioration and to preserve and/or restore function.

EPIDEMIOLOGY

The incidence of spinal cord injury in various developed countries throughout the world is estimated to be anywhere from 11.5 to 53.4 cases per million population.[8] In the United States, data from the National Cord Injury Database estimates the prevalence of spinal cord injury to be between 183,000 and 230,000 persons, or 721 to 906 per million population. Of this group, it is estimated that 21% to 28% will be found to have a syrinx if investigated between 1 and 30 years after injury, and that as many as 30% to 50% will have some degree of spinal cord cystic changes.[9] However, symptomatic syringomyelia is reported in only 1% to 9% of the spinal injury population. Syringomyelia affects mainly young persons, with an average age at presentation of younger than 29 years.[9,10] The gender distribution shows a significant male predominance, which is a reflection of the gender distribution observed for spinal cord injury.

Opinions differ regarding the relative incidence of syringomyelia when comparing complete versus incomplete spinal cord injuries and paraplegics versus quadriplegics. Rossier et al.[11] reported that in a series of 951 patients with spinal cord injuries who presented during an 11-year span, 30 (3.2%) developed syringomyelia. The authors noted a much higher incidence in patients with complete quadriplegia versus other patients. Other authors, however, failed to find any correlation between severity or location of injury and incidence of syringomyelia.[12,13]

PATHOLOGIC FINDINGS

Post-traumatic syringes are included in the general category of noncommunicating, extracanalicular syringes based on the classification scheme presented by Milhorat.[14] Syringes of this type are commonly observed after trauma, infarction, hemorrhage, and transverse myelitis. They occur as cavities separate from the central canal in vascular watershed areas, mainly in the central and dorsolateral gray matter, with a much smaller percentage occurring in white matter alone. The cavitations do not communicate with the fourth ventricle.

Unlike central canal dilations, that are lined by ependymal cells, post-traumatic syringes are lined by gliosis, which
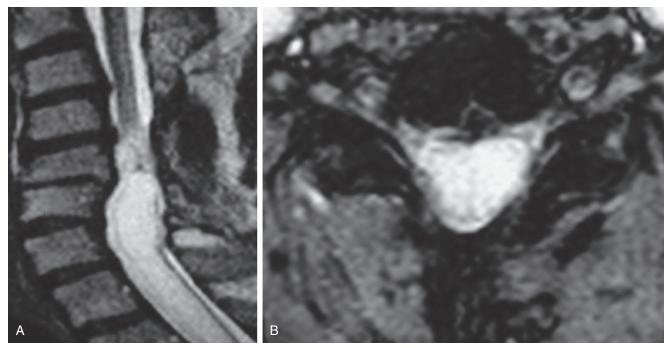
Fig. 62-1 Sagittal *(A)* and axial *(B)* view T2-weighted magnetic resonance images show a cystic cavity filled with CSF, consistent with a post-traumatic syrinx.

replaces damaged neuronal cell bodies and nerve fibers. Microglia and hemosiderin-laden macrophages are prevalent, and widening of the perivascular spaces occasionally is observed.[9] Post-traumatic syringes might communicate with the subarachnoid space via the ventromedian fissure or the dorsal–root entry zone. They originate at or near the injury site and extend rostral in 81%, caudal in 4%, or in both directions in 15%.[9,10,15] Fluid analysis reveals a composition similar to that of cerebrospinal fluid (CSF), although several studies report an increased protein content compared with that of CSF.[11,16]

PATHOPHYSIOLOGY

The trauma initiating the sequence of events that lead up to the formation of a post-traumatic syrinx has been characterized in four different scenarios, originally elucidated by Barnett and Jousse[17] and further described by other authors.[11,18] Post-traumatic syringomyelia can result from the following conditions: (1) after trauma that precipitates symptoms in a clinically silent, undiagnosed syrinx, (2) after severe trauma causing paraplegia or quadriplegia, (3) after minor trauma causing mild to moderate spinal cord injury, and (4) as a late sequela to spinal trauma causing adhesive arachnoiditis. The first scenario is unique in that it is specific to patients with congenital predisposition, such as dysraphic closure of the neural tube or obstruction of the outlet of the fourth ventricle. This leads to hydromyelia, that after repeated microtrauma, eventually progresses into syringohydromye-

lia.[18] This situation is rare; the majority of post-traumatic syringes do not communicate with the central canal.

The mechanism of formation of a post-traumatic syrinx continues to be the subject of debate. Generally, theories propose that degeneration of the initial lesion leads to cavity formation by way of hemorrhagic necrosis of cord substance, liquefaction of a hematoma, or ischemia resulting from vascular injury or arachnoiditis leading to microinfarcts and tissue remodeling.[19,20] Laboratory experiments have helped to further delineate this process; parenchymal syrinx cavities, isolated from the central canal, were reproduced with the injection of quisqualic acid, a potent agonist of several excitatory amino acid receptors, into the spinal cord substance of rats.[21,22] These results implicate excitotoxic cell death, from excitatory amino acids, as a key mechanism in post-traumatic syrinx formation.

Despite showing a clear correlation between quisqualic acid injection and cavity formation, these experiments failed to recreate the long, multilevel syringes characteristically seen in cases of human post-traumatic syringomyelia. This phenomenon was reproduced in laboratory animals treated with subarachnoid injection of kaolin, a substance known to produce arachnoiditis and a subsequent subarachnoid block, in addition to the intraparenchymal quisqualic acid injection. Animals treated solely with subarachnoid kaolin, without mechanical or chemical cord injury, however, failed to form syringes.[21,23] The conclusion is that excitatory cell death and arachnoiditis combine to cause the formation and expansion, respectively, of post-traumatic syringes.

Arachnoiditis associated with spinal cord injury leads to tethering of the spinal cord to the dural sac in the areas of scarring and to severe CSF flow disruption. A resulting pressure dissociation between the upper and lower CSF compartments is created, and distensile pulse pressure waves, occurring during Valsalva maneuvers such as coughing or sneezing, are transmitted to the CSF spaces via the epidural veins.[24] An inflow of CSF occurs via the Virchow-Robin spaces or the dorsal root entry zone and reaches the syrinx cavity via transmural fluid migration, with subsequent expansion of the cyst.[24–26]

CLINICAL PRESENTATION

Symptomatic post-traumatic syringomyelia has presented from 3 months to 34 years after initial injury.[9,11,14,15,19] Some authors have found a quicker onset of symptoms with complete cord lesions versus partial lesions, whereas others have failed to find any correlation between severity of injury and time of presentation.[2,11,13,17,27]

The most common presenting symptom in patients with post-traumatic syringomyelia is pain. It can vary in nature and location from dull, aching, constant, and cervical to burning, intermittent, and appendicular. It usually is ascending from the level of injury and, less often, can be descending. The pain often is exacerbated by coughing, sneezing, or straining. The second most common complaint is sensory loss, which usually is ascending and dissociated with loss of pain and temperature perception and relative sparing of light touch and proprioception. The third most common presenting symptom is motor weakness, which manifests as ascending weakness above the level of injury. Autonomic dysreflexia and increased spasticity are also common findings. Spasticity has sometimes been the only indicator of an expanding syrinx in an otherwise asymptomatic patient.[11,19]

DIAGNOSIS

Before the advent of magnetic resonance imaging (MRI), radiographic diagnosis of syringomyelia was painstaking and risky and involved gas myelography, which was subsequently replaced by computed tomographic (CT) myelography. Because of its noninvasiveness and its higher sensitivity, MRI has now become the diagnostic tool of choice in the workup of syringomyelia. T1- and T2-weighted axial and sagittal view images, with and without gadolinium enhancement, provide detailed information regarding the rostrocaudal and cross-sectional extent of the syrinx, septations within the syrinx, and areas of arachnoiditis and spinal cord tethering to the dura.

More specialized MRI sequences can further refine the diagnosis of syringomyelia. Cine flow (Fig. 62-2) studies can reveal areas of CSF flow interruption and confirm regions of adhesive arachnoiditis. Animal experiments also have shown that diffusion-weighted images detect post-injury cystic cavi-

tation of the spinal cord sooner than do conventional T1- and T2-weighted images and as early as 1 week after injury.[22]

Alternatively, for those patients who cannot safely undergo MRI, CT myelography remains the next best option. After the intrathecal administration of a water-soluble contrast agent, delayed CT scans are obtained 4 to 24 hours later, which allows time for the contrast agent to enter the cyst, that is then detected by the CT scan.[24]

TREATMENT

A few series document the conservative treatment or observation of certain patients with post-traumatic syringomyelia, some of whom had refused surgery. Barnett and Jousse[7,17] described a state of "quadriplegic helplessness" that eventually develops in these patients. Although a number of other authors failed to see patients' progress to this state of decline, and some actually reported stabilization or, rarely, resolution of symptoms, the majority of these patients who were managed nonoperatively showed a gradual worsening of symptoms or the development of new symptoms.[11,19,27] The general consensus is that all patients with neurologic deterioration and evidence of a syrinx should be managed surgically.

As early as 1898, syringotomy was proposed as a treatment for syringomyelia by Abbey, who performed the procedure on an autopsy subject.[2,28] In the late 1950s, the experimental results achieved by Freeman[29] and by Freeman and Wright[30] convinced them to manage post-traumatic syringomyelia in humans with syringostomy. Initially, the goal was marsupialization of the syrinx. With advances in shunt material, shunting of the syrinx to the subarachnoid, pleural, and peritoneal spaces has become a cornerstone in the surgical management of post-traumatic syringomyelia.

In addition to shunting of the syrinx, our surgical approach addresses the presumed causative factor that promotes syrinx propagation: arachnoiditis and interruption of CSF flow (Fig. 62-3). We monitor our patients throughout the operation with somatosensory-evoked and motor-evoked potentials to aid our intraoperative decision making and to guide our neuroprotective measures, such as hypothermia and intravenously administered steroids. A laminectomy is performed at the level corresponding to the epicenter of the syrinx and over sites of spinal cord tethering. Intraoperative ultrasonography is performed to confirm adequate dural exposure (Fig. 62-4). The dura is opened away from any areas of tethering to avoid spinal cord injury, and sites of spinal cord and nerve root tethering to the dura are meticulously released. Untethering alone often is enough to collapse the syrinx.[31] Repeat ultrasonography is next performed to check for collapse of the syrinx, free flow of CSF around the cord, and pulsation of the cord. If the cyst has collapsed with untethering alone, expansile duraplasty is performed with cadaveric dural allograft sewn in a running, watertight fashion with 5-0 Prolene suture. The closure is confirmed to be
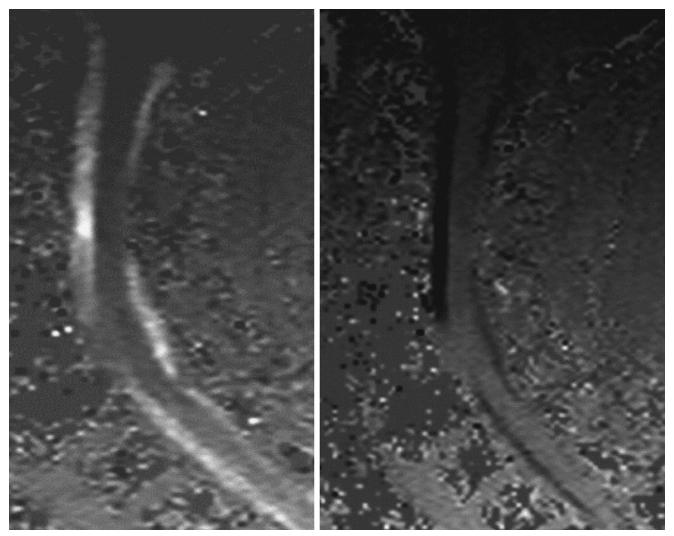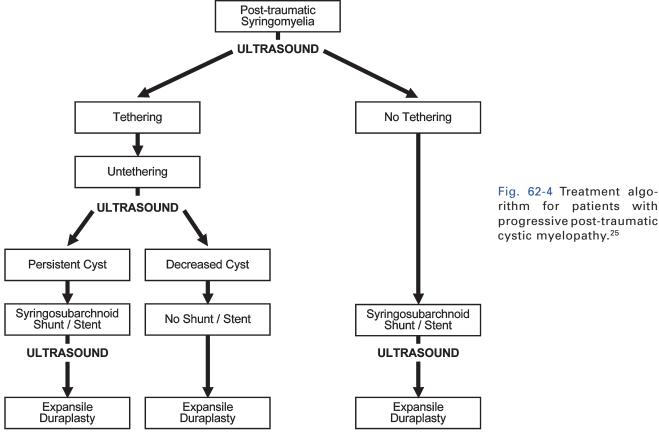
Fig. 62-2 Cine magnetic resonance images show the dual direction of flow of CSF around the spinal cord. Obstruction of CSF flow at the level of spinal cord tethering can be seen.

Fig. 62-3 Sagittal intraoperative ultrasonographic view of the lower thoracic spinal cord shows a large syrinx.

watertight with repeat Valsalva maneuvers. The duraplasty affords a more physiologic state of CSF flow around the cord, free of adhesions and tethering. Postoperatively, patients are kept on bedrest for 5 to 7 days, sometimes with a lumbar drain if dural closure was not satisfactory, to allow the duraplasty to heal and decrease the risk of postoperative pseudo-meningocele formation.

If the syrinx persists despite untethering, we proceed with shunting. An approximately 2-mm midline myelotomy is made at the caudal end of the syrinx with a No. 11 blade. A Spetzler silastic microcatheter is then advanced rostrally into the syrinx cavity as far as the cavity will allow. Approximately 1 cm of catheter is left outside the substance of the cord and is positioned in a region free of adhesions to prevent outlet obstruction. Ultrasonography is repeated to evaluate positioning of the catheter, assess for collapse of the syrinx, and ensure adequate untethering and good pulsations of the cord. The

```
                    Post-traumatic
                     Syringomyelia

                      ULTRASOUND

         Tethering                      No Tethering

        Untethering

        ULTRASOUND

  Persistent Cyst    Decreased Cyst

 Syringosubarchnoid  No Shunt / Stent   Syringosubarchnoid
   Shunt / Stent                          Shunt / Stent

   ULTRASOUND                             ULTRASOUND

   Expansile          Expansile           Expansile
   Duraplasty         Duraplasty          Duraplasty
```

Fig. 62-4 Treatment algorithm for patients with progressive post-traumatic cystic myelopathy.[25]

catheter is then secured to the pial surface using a 7-0 Prolene suture. The duraplasty is next performed as described above. Meticulous hemostasis throughout the procedure is critical to prevent blood from entering the subarachnoid compartment and to prevent further arachnoid scarring.

Other currently used surgical options include cord transection at the level of the syrinx in complete paraplegics, neuroendoscopic fenestration of cyst septations, and myelopediculotomy (laser fenestrations through the cyst wall).[24] It should also be emphasized that timely reestablishment of normal spine/spinal cord anatomy by correction of kyphosis, cord compression, and spinal instability will avoid obstructions to CSF flow and decrease the chance of cyst formation. Schurch et al.[19] found complete cyst collapse in two patients after simple realignment of the spine.

OUTCOMES

Postoperative complications include shunt failure, infection, pseudomeningocele, peritoneal pseudocyst, and CSF overdrainage with headaches. Shunt failure rates have been quoted from 10% to 100%, with most series reporting rates of approximately 50%.[9] In general, postoperative complication rates are estimated anywhere from 6% to 18%.[9]

Although long-term follow-up will be needed before solid statements regarding the results of surgical treatment can be made, most studies show a good correlation between syrinx decompression and symptomatic improvement.[24] During the short-term postoperative period, most patients show improvements in pain and in motor deficits more commonly than in reversal of sensory deficits.

CONCLUSION

Post-traumatic syringomyelia has been recognized as a pathologic and clinical entity for many decades. The syringes normally occur in watershed areas, such as the central and dorsolateral gray matter, are lined by gliosis, and do not communicate with the central canal or fourth ventricle. Clinical and animal studies have emphasized that excitotoxic cell death and arachnoiditis are central to the formation and expansion of post-traumatic syringes. This creates a condition whereby pulse pressure waves, such as those caused by Valsalva maneuvers, are transmitted to the subarachnoid compartment by the epidural veins, resulting in the transmural migration of CSF into the cyst cavity.

Most commonly, patients present with pain, although ascending sensory loss and motor weakness also are common complaints. MRI is the diagnostic tool of choice. It accurately delineates the morphology and extent of the

syrinx. Surgery should be aimed at restoring normal CSF flow dynamics and ultimate cyst collapse via shunting. Good outcome normally correlates with syrinx decompression, and most patients show improvement in preoperative symptoms.

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Rehabilitation and Recovery after Spinal Cord Injury

INTRODUCTION

Spinal cord injury (SCI) is one of the most devastating injuries a person can sustain. Of paramount concern to patients and their families is the degree of return of function they can expect. This chapter reviews the general recovery and rehabilitation issues associated with motor function recovery and other activities of daily living after SCI.

The incidence of SCI has been estimated to be 15 to 40 cases per million population worldwide, with the United States at the high end of those estimates.[1] Considering the current size of the United States population, this translates to approximately 12,000 new cases of SCI each year. Approximately 4000 people die before reaching a hospital, and approximately 1000 patients die during hospitalization.[1] Prevalence of SCI in the United States is now approaching 250,000 people and is increasing as a result of increasing survival rates.[2,3] After sustaining traumatic SCI, the patient's neurologic baseline is uniformly established based on the neurologic classification system of the American Spinal Injury Association (ASIA) (Fig. 63-1). It allows for effective communication and monitoring of the patient among different physicians, disciplines, and organizations. Rehabilitation efforts ideally are started from the time that the patient's SCI severity has been established and are performed by a team. In addition to attention for neurologic recovery, use of residual function should be optimized, and this process often is started immediately during the perioperative period in the hospital.

LEVEL OF INJURY

Terminology has been consistent throughout disciplines, largely because of excellent efforts by the ASIA.[4] Based on their classification system, we speak of *tetraplegia* or *quadri-*

plegia if SCI causes compromised functioning of upper limbs, trunk, lower limbs, and pelvic organs because of injury at the level of the cervical spine. *Paraplegia* refers to damage of the spinal cord below the cervical segments with sparing of upper extremity function. An injury is *complete* when there is no sensory or motor function preserved in the lower sacral segments; *incomplete* SCI is characterized by preservation of at least some motor and/or sensory function below the damaged level of the spinal cord with preserved S4-S5 root function. To determine the level and grade of injury according to the ASIA impairment scale (see Fig. 63-1), the lowest level of bilateral motor function is evaluated. Muscle strength grade 3 or 4 is considered normal, provided that the level immediately proximal to this has 5/5 strength (grade 5 = full strength).

The level of sensation is evaluated by using both sharp and light touch in specific dermatomes, as outlined in Figure 63-1. Although light touch might be perceived, sensation is graded as absent if the pinprick does not provoke a sharp sensation. The level is determined bilaterally based on the last dermatome in which the patient adequately identified sharp sensation. Documentation of the motor and sensory levels is of vital importance for monitoring the stability and rehabilitation progress of the patient.

PROGNOSIS

Although families obviously are anxious to learn about the prognosis, prognostication usually is more accurate 72 hours after initial injury.[5] At that point, ASIA grade A injuries rarely become incomplete. To the contrary, ASIA grade B injuries in patients who exhibit preservation of pin prick sensation have an excellent prognosis to become ASIA grade D or even E injuries.[6,7] Without preservation of sharp sensation, injuries rarely progress to ASIA grade C or D.[8] Also, injuries evaluated as ASIA grade A at the emergency department that subsequently become incomplete rarely progress to ASIA grade C or D. In rare cases of patients with ASIA grade A injuries who walk a few months after injury, the initial grading probably was too pessimistic and careful examination at the emergency department most likely would have shown an element of sacral sparing. Recovery rates of

one to two levels have been reported as being up to 90% and 66%, respectively, approximately 18 months after injury in tetraplegic patients, except those with C4 level injury.[9,10]

Patients who are tetraplegic but have preserved pin prick sensation in the lower extremities have an excellent chance of walking approximately 1 year after injury in approximately 67% of cases.[7] In the situation of a motor incomplete examination after 72 hours, patients have been reported to walk in 87% of cases after 1 year.[18] These results depend on the level of initial injury.

REHABILITATION

As stated previously, the rehabilitation process ideally should start at the time the patient enters an SCI facility because a considerable amount of neurologic recovery usually is seen within the first few months after SCI.[11] At most institutions, SCI rehabilitation has multiple phases, starting with an evaluation and stabilization phase. The level and degree of injury are established based on the ASIA impairment scale, and medical and surgical treatments are initiated. Skeletal stability is paramount and often necessitates spinal decompression and fusion. During the second phase, medical complications need to be addressed immediately postoperatively and the patient usually is placed in an orthotic device to stabilize the spine. The third or subacute phase of SCI rehabilitation can be limited by concurrent medical problems, healing fractures of the extremities, autonomic dysfunction, and pressure ulcer formation. Necessary bracing and splinting can also limit progress at that time.

Neurologic recovery and prognosis often are reviewed, and close communication with the patient and family is maintained. Increasing evidence has accumulated that it is best to perform skeletal stabilization procedures sooner rather than later to decrease time spent in the intensive care unit and facilitate early discharge to a rehabilitation facility to improve functional recovery.[11] Therapy begins at the bedside in the acute care facility to prevent contractures (splinting and range of motion), and muscle strength is maintained as much as possible. Pressure ulcer prevention is vital, and education to the patient and supportive family plays a significant role. Nutritional issues have to be addressed, and deep venous thrombosis (DVT) prophylaxis is initiated immediately to prevent deadly pulmonary emboli.[12,13] This often includes placement of a vena cava inferior filter to prevent proximal migration of emboli from the lower extremities or pelvic veins. In the pediatric trauma population, the incidence of DVT and pulmonary embolism (PE) is fortunately much lower, but it should never be overlooked as a possible cause of sudden patient compromise.[14]

As soon as the patient is medically stable, discharge to a rehabilitation facility should be initiated, although when a patient is unable to participate with full rehabilitation, an interim stay at a nursing facility might be required. The average length of stay in a rehabilitation facility in the United States is estimated to be 60 days (±38.7 days) after SCI, which is the shortest in the world.[15] Cultural differences probably are responsible for inpatient stays up to 250 days in countries including Japan, The Netherlands, and Norway.[16] Shorter stays in rehabilitation facilities historically are associated with high levels of functional ability, less severe injuries, and strong

ASIA IMPAIRMENT SCALE

☐ **A = Complete:** No motor or sensory function is preserved in the sacral segments S4-S5.

☐ **B = Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.

☐ **C = Incomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.

☐ **D = Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.

☐ **E = Normal:** Motor and sensory function are normal.

CLINICAL SYNDROMES

☐ Central Cord
☐ Brown-Sequard
☐ Anterior Cord
☐ Conus Medullaris
☐ Cauda Equina

A

Continued

Fig. 63-1 The American Spinal Injury Association (ASIA) *(A) and* impairment scale *(B).*
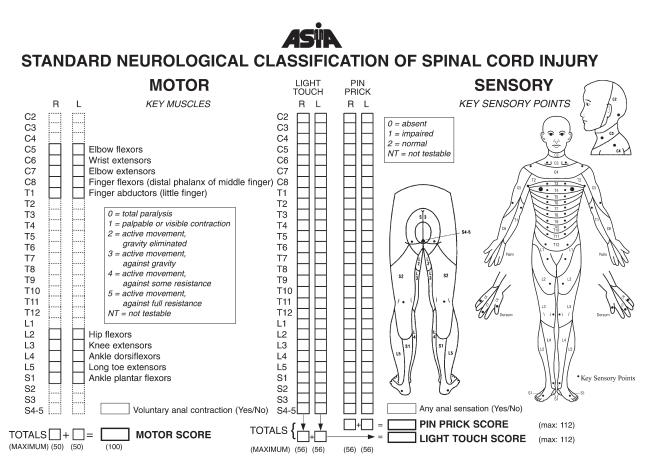
Fig. 63-1, cont'd *B*, The American Spinal Injury Association neurological classification of spinal cord injury.

social support.[15,16] The patient's ultimate functional outcome generally depends on the level of injury and degree of neurologic impairment. Depending on the patient's motivation, age, body habitus, and general state of health, levels of functioning might differ. Table 63-1 contains the typical functional outcomes for patients with complete SCI.[17]

MEDICAL COMPLICATIONS

SCI compromises not only motor function and sensation but every major organ function in the body. This leads to a significant number of rehospitalizations despite improvements in SCI medical management. Urinary tract infection (UTI) remains the leading cause for readmission. Additionally, tetraplegic patients most often are admitted for respiratory issues whereas paraplegic patients are more likely to suffer from pressure ulcers for which readmission is required. Lower functional recovery levels (FIM) are significantly predictive of rehospitalization.[19] The details are discussed in the following.

NEUROGENIC BLADDER

Manifestations of a neurogenic bladder include the loss of sensation of fullness and the inability to voluntarily initiate urination and completely empty the bladder once urine is flowing. This results in incontinence and urinary retention, reasons why bacterial colonization of the bladder occurs in approximately 80% of patients with SCI. UTI is common and remains the number one cause for readmission to a hospital.[19] The level of SCI is predictive for the development of neurogenic bladder, and the reason lies in the compromised functioning of the autonomic nervous system. Because of co-contraction of the bladder wall and the bladder outlet, urination does not initiate and emptying of the bladder is impossible against a closed sphincter near the end of voiding. This is called detrusor-sphincter dyssynergia. Patients with lesions below the spinal cord (T12-L1) in the cauda equina tend to have a flaccid, areflexic bladder.

Clean intermittent catheterization is a treatment option for neurogenic bladder, as is a condom or indwelling catheter. Spontaneous voiding also is an option, although it is difficult

TABLE **63-1** Typical Functional Outcomes for Patients with Complete SCIs

LEVEL OF INJURY	PRESSURE RELIEF	WC TRANSFER	WC PROPULSION	AMBULATION	ORTHOTIC DEVICES	TRANSPORATION
C3-C4	Dependent in bed or in WC. Independent in power recliner	Complete dependence	Power WC independent with chin or pneumatic control	Not applicable	Upper extremity externally powered	Dependent on others
C5	Most require assistance	One assistant	Independent power WC. Short distances adapted manual WC		Upper extremity externally powered	Independent driving in adapted VAN
C6	Independent	Possible independent with transfer board	Independent manual WC for moderate distances. Needs assistance outdoors		Wrist driven orthoses	Independent driving in adapted VAN
C7		Independent, also into car. Dependent onto and from floor	Independent indoors and outdoors except stairs		None	Independent driving with handheld controls, independent with placing WC in car
C8-T1		Independent also onto and from floor	Independent indoors and outdoors. Assistance with stairs	Exercise only, requires assistance/guarding		
T2-T10		Independent	Independent	Exercise only may not require assistance	KAFO with forearm crutches	
T11-L2				Functional ambulation indoors with orthoses	KAFO with forearm crutches	
L3-S3				Community ambulation indoors and outdoors with orthoses	AFO with crutches or cane	

WC, wheelchair; KAFO, knee-ankle-foot-orthosis; AFO, ankle-foot-orthosis.
Adapted from Delisa JA (ed): Rehabilitation Medicine: Principles and Practice, 4th ed. Philadelphia, Lippincott Wilkins & Williams, 2004.

to achieve. Discussions regarding bladder management have to include the patient's dexterity and hand function, as well as urodynamics and the voiding pressures associated with the neurogenic bladder. In the pediatric population, clean intermittent catheterization can be started as young as 3 years of age to provide a regular, predictable, and complete emptying of the bladder while mimicking "normal" urination after filling for a set period of time. This has been shown to improve reaching continence, and it reduces the risk of UTI and renal complications.[20] Self-catheterization can be taught to a 5- to 7-year-old child if he or she is mentally and functionally stable. The child must be able to carefully follow a step-by-step process and has to be able to tell time.[20] Continence is expected only when children reach school age, but it is essential for adolescents to move on to the challenges of living an independent and satisfying adult life.

NEUROGENIC BOWEL

Management of bowel emptying can be a lifelong struggle for patients with SCI. The ultimate goals are continence and timed, regular evacuation. The method for achieving these goals is different for every patient but usually requires three different types of stool softeners administered throughout the day. Some patients require or even prefer manual disimpaction; others use frequent enemas, mainly in the evening. Colostomy is only rarely indicated in a patient with SCI.

Pediatric patients can start a bowel program usually at the age of 3 years, with the main goal being to prevent constipation. Toddlers can be placed on a potty chair or toilet at regular intervals and taught to "bear down" to successfully achieve regularity. Although the desire for privacy for bowel management might increase, parents have to remain available for their

growing child because complications can be serious if problems such as constipation or ulcer are not identified early.[21]

AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia is a life-threatening, uninhibited sympathetic nervous system response to noxious stimuli below the level of injury, more often observed in patients with T6 or higher lesions. It has been shown to affect up to 85% of patients after higher level SCI and can affect them more than once in a lifetime.[22,23] It typically is manifested by a pounding headache, flushing and profuse sweating above the level of injury, piloerection, blurred vision, and nasal congestion and often comes about when patients recover from spinal shock. More often, however, it begins to occur 2 months after initial SCI.

The cause for the imbalance is the splanchnic outflow from T6 to L2.[24] Noxious sensory stimulation below T6 ascends in the sympathetic intermediolateral gray matter, leading to reflex sympathetic discharges. Unopposed release of norepinephrine and dopamine occurs because inhibitory signals from the brain are being blocked because of the SCI. The most common causes are bladder overfilling, bowel obstruction, pressure ulcers, and fractures.[25,26] Communication is essential, especially with children, and includes education on a consistent bladder and bowel program to the patient, parents, and community members who have contact with the child.[21]

The most important task for health care workers is to find the source of noxious stimulation. Meanwhile, the blood pressure must be monitored often because hypertension up to 300 mm Hg has been reported. The head of the patient must be elevated. If no obvious causes can be discovered quickly (e.g., kinking of an indwelling catheter or impaction) and the blood pressure remains high, pharmacologic agents such as nitroglycerin or nifedipine can be used.[24] Surgeons also inflict noxious stimuli when performing interventions below the T6 level on a patient with high-level SCI. Although the patient might not feel pain, anesthesia and/or close monitoring is essential to prevent autonomic dysreflexia from taking place.

DEEP VEIN THROMBOSIS AND PULMONARY EMBOLISM

DVT and PE are potentially lethal complications of SCI and occur in a substantial number of patients. Death from a PE accounts for 8% of all causes of death after SCI, and DVT has been reported to occur in up to 100% of patients with SCI.[27] The causes for such a high rate of DVT lie in Virchow triad: bed rest, hypercoagulability, and paralysis. Pneumatic compression devices, anticoagulants, and aggressive monitoring of the patient immediately after SCI are ways to prevent and identify clots early. The compression boots should be left in place for the first 2 weeks after injury, combined with some type of pharmacologic DVT prophylaxis. After review-

ing the available data, low–molecular weight heparin, adjusted dose heparin, or anticoagulation in conjunction with pneumatic compression devices is a better alternative to low-dose heparin therapy alone.[27] Inferior vena cava filters also are efficacious for the prevention of PE in patients with SCI; however, efficacy of inferior vena cava filters versus prophylactic combination therapy with low–molecular weight heparin and pneumatic compression devices has not been studied. Very few thrombolic events occur beyond 3 months from the time of SCI, and only if the patient is at high risk should anticoagulation be continued after that time. The risk for bleeding complications simply outweighs the risk for the development of DVT. Therapy can be discontinued earlier if lower-extremity motor function is regained. For symptomatic patients, duplex ultrasonography is an effective and noninvasive study to determine the presence of DVT with sensitivities of approximately 90%. The gold standard but invasive venography can be reserved for patients who are at high risk or for cases in which clinical suspicion is high and the ultrasonographic findings are negative.

PULMONARY COMPLICATIONS

Because the diaphragm is a critical muscle for respiration, innervated by the phrenic nerve from C3, C4, and C5, paralysis can lead to atelectases, pneumonia, and failure of ventilation. Fortunately, the majority of patients admitted on a ventilator can be weaned off, including approximately half of all C3 tetraplegics.[28] Patients with lower level SCI have reduced function of the accessory respiratory muscles (interscalene, C5-C8) and intercostal muscles (T1-T11). Forced expiration and coughing largely are performed via abdominal muscles innervated from T6 through T12. Even if the diaphragm is intact, patients with SCI have vital lung capacities of approximately 30% of normal. Because the body stabilizes over time, this improves to approximately 60%. Pulmonary toilets need to be performed consistently, including chest physical therapy, assisted coughing, and even the use of pharmacologic bronchodilation.[29]

PRESSURE ULCERS

Pressure ulcers are a major complication associated with acute and chronic SCI. Despite the incidence and prevalence, this is a preventable comorbidity as long as the pressure does not exceed a certain intensity and duration at one location on the body. Because SCI also changes the composition of the skin below the level of injury by reducing proline, lysine, and hydroxylysine concentrations, the impact of immobility is exponential. Additionally, bowel incontinence, bladder incontinence, and diminished sensation contribute to loss of skin integrity, which becomes a significant risk for systemic infection.[30] Generally, four stages of ulceration are described based on the degree of tissue damage observed.[31] Stage I is described as nonblanching erythema with intact skin,

although this often is difficult to diagnose on pigmented patients or in the presence of an eschar. Stage II involves partial skin loss of the epidermis and/or dermis. Stage III has been reached if full thickness skin loss with underlying necrosis but an intact subcutaneous fascia (the "deep" fascia) is present, whereas Stage IV additionally involves deeper destruction of muscle and bone and sometimes a sinus tract.[31]

To prevent long-term immobilization and pressure build-up, an unstable spine must be stabilized to allow a patient to get out of bed and become vertical, preferably without a brace. In addition, the nutritional status of the patient must be optimized and serum levels of albumin need to be checked. Obesity is not to be confused with good nutrition, and adipose tissue often is poorly vascularized, which contributes to the risk of developing an ulcer.[32] Smoking, fevers, and incontinence have independently been shown to contribute to skin compromise, and nursing staff need to assist the patients with more frequent position changes.[33,34]

PAIN AND SPASTICITY

Pain is experienced by a significant number of patients with SCI. It usually presents within 6 months after the injury and often compromises the patient's rehabilitation participation. In pediatric patients, pain can cause fear and works against all rehabilitation efforts. Pain occurs in at least 70% of patients with SCI.[35,36] It interferes with activities of daily living in almost half of all patients with SCI.

Pain after SCI can be classified by several systems.[37,38] Musculoskeletal pain generally is achy and confined to specific anatomic areas. Pain can occur from overuse of functional extremities, an altered gait pattern, or direct trauma or injury. Visceral pain is dull and vague and usually localized in the chest, abdomen, or pelvic area. Neuropathic pain is common and can be sharp, burning, electrical, or felt in other ways. Segmental neuropathic pain at the level of SCI can be radicular or central pain, but a peripheral nerve lesion also is possible. Neuropathic pain distal to the lesion usually is caused by central pain. Other reasons for pain include syrinx formation, headaches associated with dysreflexia, complex regional pain syndrome, and psychogenic pain.[39,40]

Treatment is critical to allow the rehabilitation process to continue, and range-of-motion and strengthening exercises are indicated. The sitting position in a wheelchair must be evaluated and adjusted, and optimal body mechanics during activities of daily living are essential. Biofeedback usually helps patients with their discomfort, and low-dose anti-inflammatory medications also can be helpful. The use of ice packs or heat warrants caution because lack of protective sensation can cause frostbite or burns. Long-term use of narcotic pain medication usually is not advocated because of additional problems and the effects on bowel routine. A transcutaneous electrical nerve stimulator unit might be helpful to some.[40]

Spasticity is defined as velocity-dependent muscle resistance during range of motion. This is caused by an upper motor neuron lesion and is a common side effect of SCI. Initially, deep tendon reflexes are depressed after SCI (spinal shock), but after a few weeks or months, reflexes can sometimes increase to severe levels. They can inhibit patients from performing activities of daily living, cause pain, and interfere with hygiene. Baclofen and tizanidine are first-line oral treatments; dantrolene and Valium usually are reserved for worsening states of spasticity because of their sedative side effects. As for patients with cerebral palsy, intrathecal baclofen can be considered, although reasonable results have been obtained with intramuscular injections of botulinum toxin.

PATHOLOGIC FRACTURES AND OSTEOPOROSIS

Because of significant changes in patients' mobility, extreme bone loss occurs during the first 4 to 6 months after SCI, below the level of the lesion. The rate of risk for fractures has been reported as high as 34%.[41] The osteopenia usually stabilizes a year and a half after injury.[42–44] The best treatment to prevent severe bone loss includes mobilization with standing activities. Diphosphonates have been shown to reduce bone resorption in this population. In case of erythema and swelling of an extremity below the level of the SCI, fracture must be suspected in addition to a DVT. Proper radiographic studies need to be obtained to prevent unnecessary administration of antibiotics for a presumed cellulitis. Fracture care is not very different from the treatments offered to patients with intact sensation and locomotion, especially because benign neglect of fractures can cause severe skin compromise and lethal infections. Although many fractures can be managed conservatively, proper splints need to be applied and alignment of healing fractures must be checked regularly.[44] Especially midshaft and proximal femoral fractures with improper alignment can cause the patient to develop significant sitting imbalance if the thigh cannot be used for support. Skin breakdown is not uncommon. If alignment is appropriate, nonunions are better tolerated and generally well accepted in nonambulatory patients.[45]

HETEROTOPIC OSSIFICATION

Heterotopic ossification occurs in more than 50% of all patients with SCI below the level of injury. The hip joint and the knees are most commonly affected. Heterotopic ossification is defined as the development of new bone around moving joints. Because of the severity of bone formation, approximately 5% of patients end up with significant joint stiffness, nerve entrapment, or pressure underneath the skin.[45] In those cases, the ectopic bone must be removed to prevent skin compromise or pain.[46] Aggressive physical therapy can sometimes keep a joint mobile, assisted by oral diphosphonates if started early enough. When the skin is erythematous and warm, radiographic evaluation must rule

out fracture and ectopic bone before diagnosis of DVT or cellulitis. Radiation therapy and indomethacin have been shown to be effective for patients without SCI, although efficacy in this patient population has not been proven.

Surgery to remove the bone can be complicated by heavy bleeding, especially if the heterotopic ossification has not reached its final stage. Once burnt out, it usually can be removed safely in cases of ankylosed hips or knees. Diphosphonates or radiation can be used to prevent recurrence postoperatively,[47] although little evidence of their efficacy exists.[48]

ACKNOWLEDGMENT

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